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FOR "PERHAPS THE MOST COMMON DEFICIENCY"

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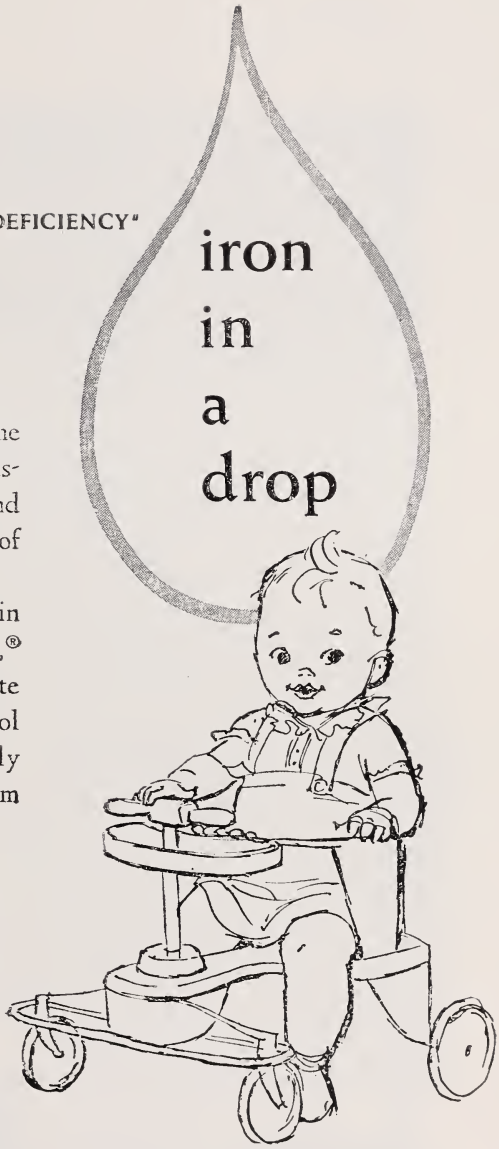

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(1) Youmans, J. B., in Handbook of Nutrition, Chicago, American Medical Association, 1951, p. 577; (2) Hansen, A. E., in Mitchell-Nelson Textbook of Pediatrics, ed. 5, Philadelphia, W. B. Saunders Co., 1950, p. 106; (3) Heck, F. J.: J.A.M.A. 148: 783, 1952.

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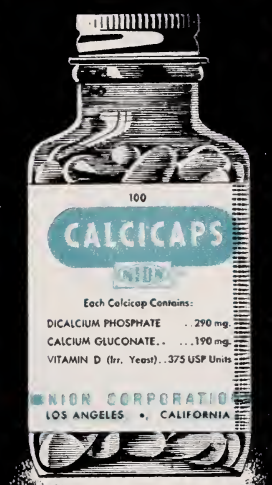

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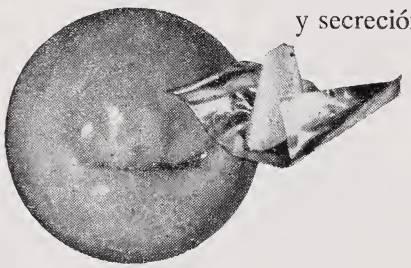

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†Steinberg, C. L., and Roodenburg, A. L.: J.A.M.A. 149:1458, 1952.

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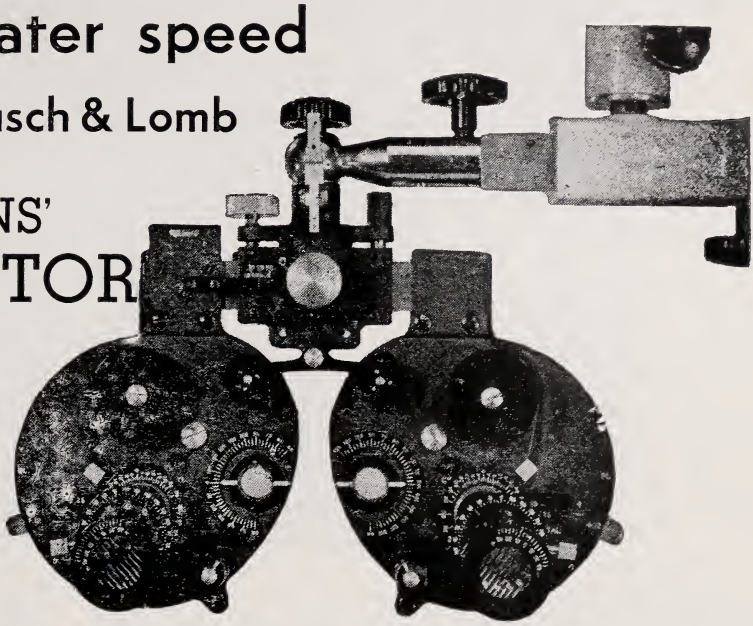

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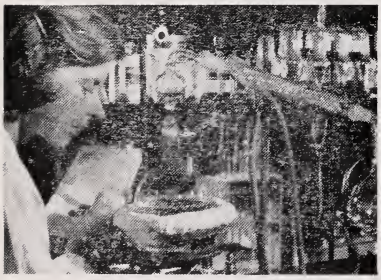

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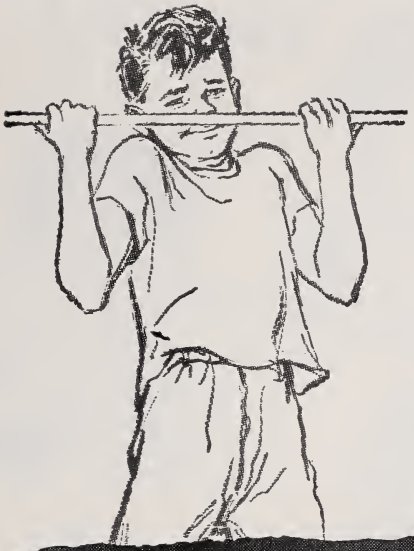

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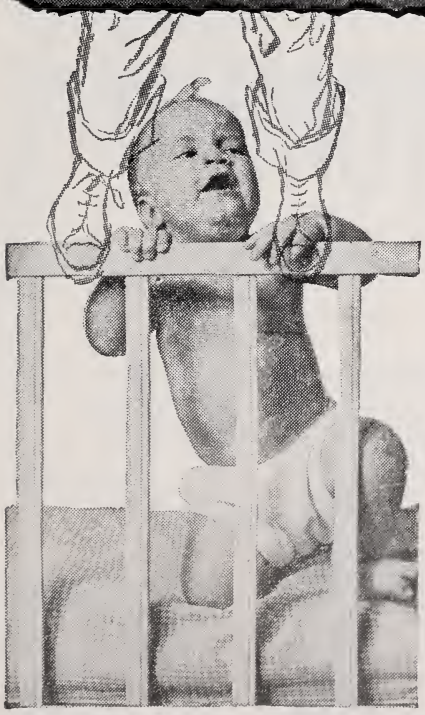

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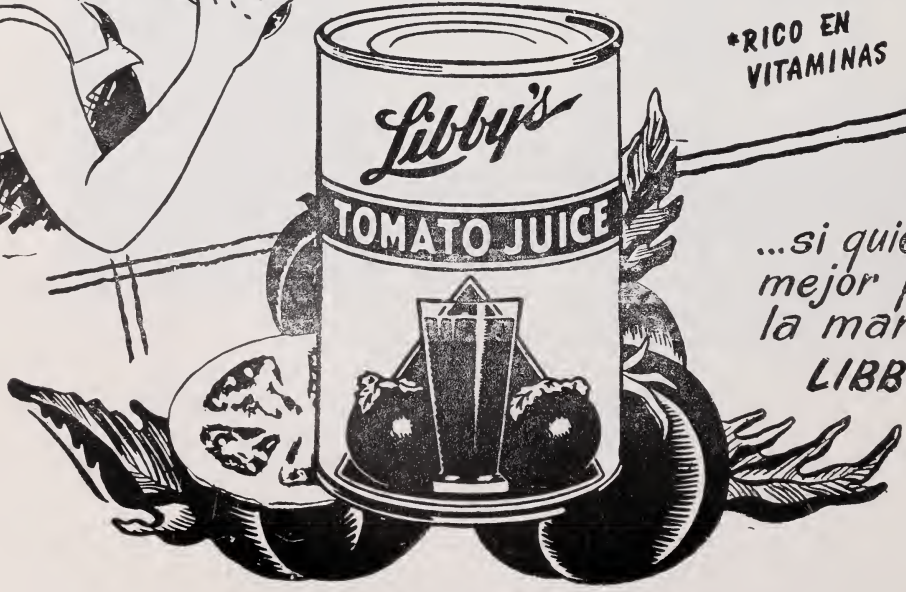

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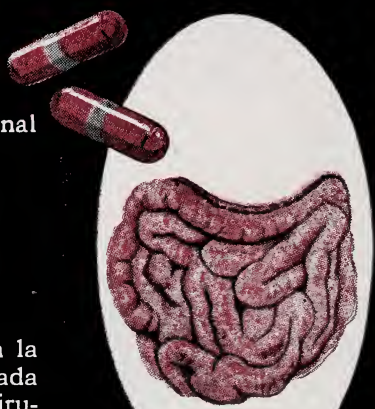

INDICACIONES: Infecciones entéricas de tipo diarreico

Infecciones mixtas en cirugía intestinal

Tuberculosis anorrectal

Fístula anal

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BOLETIN
DE LA
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VIRAL HEPATITIS IN THE PUERTO RICAN MALE VETERAN

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Introduction: Although it appears that viral hepatitis, as it occurs in Puerto Rico, is similar to the one described elsewhere,[1, 2] to our knowledge, this impression has not been substantiated in the Puerto Rican literature with a sizeable series of cases. For this reason, and being aware of the relative frequency with which this clinical entity was met at the San Patricio VA Hospital during the last six years, it was decided to gather all the available records, analyze the clinical and laboratory aspects of the disease, and publish our findings. The cases that were accepted for analysis were those in which the clinical and laboratory information that supported the diagnosis, was ample, adequate and definite. To the best of our ability all forms of toxic and parasitic hepatitis, Weil's disease, and infectious mononucleosis, have been excluded. Sixty eight cases were gathered, fourteen of which fell into the category of homologous serum hepatitis.

Infectious Hepatitis: Fifty-four cases of infectious hepatitis were collected from the files of the San Patricio VA Hospital through the years 1947 to 1953.

Age: The age at which this disease was encountered ranged from 21 to 75 years. However, as it appears from Table I, infectious hepatitis tends to occur with greater frequency in the younger age groups. Of course, this does not represent an overall age incidence, for children and females are not included in our study. Never-

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TABLE I

AGE INCIDENCE OF INFECTIOUS HEPATITIS

Age	Number of Cases	Per Cent of Cases
20-29	28	52
30-39	17	32
40-49	3	5
50-59	1	2
60-69	3	5
70-75	2	4

theless, our findings suggest that at least in the Puerto Rican male veteran, this disease is not common after 40 years of age.

Signs and Symptoms: From Table II, one can observe the frequency with which various signs and symptoms of the disease were found. Jaundice appears to be almost a sine qua non for the diagnosis of infectious hepatitis. There were only two cases described as anicteric, but actually only one could be accepted as such, for the other was admitted to the hospital at a time when he was

TABLE II

OVERALL INCIDENCE OF SIGNS AND SYMPTOMS IN 54 CASES OF INFECTIOUS HEPATITIS

Signs or Symptoms	Number of Cases	Per Cent
Jaundice	52	96
Anorexia	50	92
Malaise and/or lassitude	50	92
Dark urine	47	87
RUQ tenderness	45	83
RUQ pain	41	74
Loss of weight	37	70
Fever	31	57
Nausea and/or vomiting	26	48
Light or clay colored stools	25	46
Itching	14	26
Diarrhea	9	17
Spider angiomata	3	5
Palpable spleen	1	2

recovering from his illness and conceivably could have been icteric prior to admission. It is of interest to note that 25 cases had light or clay colored stools and 14 had itching. In all the cases where itching occurred, it was transient, lasting only a few days. The question as to why itching occurs in liver disease still remains unanswered, as there was no definite relationship of its development to the bilirubin content, cholesterol ester fraction or to the duration of the disease or of jaundice. In nine cases with pruritus where a serum alkaline phosphatase was determined, an abnormally high value was obtained. However, there were 16 cases with high alkaline phosphatase who did not exhibit itching. Twenty five of 36 cases where specific reference was made to stool description, had light colored or acholic stools. Of these, only ten had itching. Four other cases had itching but no reference was made in relation to the stools. As reported elsewhere,[1,2] diarrhea was not a prominent symptom. Though spider angiomata have been reported to occur in infectious hepatitis,[1] in the three instances where they were present in our series, the evidence favors the existence of a concomittant cirrhosis of the liver, either prior to the hepatitis or subsequently as a sequela.

Prodromata consisting mainly of malaise, lassitude, anorexia, and fever were present in the majority of the patients. In 48 cases the initial symptoms could be determined from the information written in the record. Anorexia and malaise by themselves, or either of them combined with another of the symptoms listed in Table II constituted the most frequent group of symptoms at the

TABLE III

INITIAL SYMPTOMS IN ORDER OF FREQUENCY IN 48 CASES*

Signs or Symptoms	Number of Cases	Per Cent
Malaise and/or lassitude	27	56
Anorexia	25	52
Jaundice	16	33
RUQ pain	15	31
Dark urine	14	29
Fever	13	27
Nausea and/or vomiting	10	20
Diarrhea	3	6
Loss of weight	2	4
RUQ tenderness	42 (on admission)	

* In 6 cases the initial symptom could not be determined.

onset of illness. However, by the end of the first week, jaundice, dark urine, right upper quadrant pain, and tenderness associated with hepatomegaly, had also made their appearance. With the exception of itching, all the signs and symptoms listed in Tables II and III appeared before the end of the second week of illness. Though we can not adequately ascertain the onset of right upper quadrant tenderness, at least we can state that it was a prominent finding on admission. Thirteen cases showed acholic stools sometime during their first week of illness. Of 16 cases where jaundice was the initial symptom, no reference was made as to the time of appearance of dark urine in six. In four cases jaundice appeared before, and in the remaining six, simultaneously with dark urine. All of the cases (47) in which specific reference was made to the presence of dark urine, exhibited it. Of these, eight patients noticed dark urine prior to jaundice, and four after jaundice was observed. The appearance of dark urine prior to the onset of jaundice seems to be of common occurrence.[1, 3].

Disappearance of symptoms: As a rule the symptoms that appeared first, disappeared first with the exception of jaundice, which was the initial symptom in 16 cases and was the first to disappear in only one instance. In addition to jaundice, the other finding which disappeared last on six occasions, was tenderness over the right upper quadrant. With regard to the disappearance of dark urine and acholic stools, we do not have specific information in number of days, however, it is our impression, obtained from occasional statements written in the records, that the presence of acholic stools is of short duration, while that of dark urine lasted much longer. Of prognostic value appears to be the following: when symptoms such as fever, anorexia, malaise, and lassitude, persisted for more than two weeks, the case as a rule turned out to be a moderately severe or severe one, from the viewpoint of duration of disease, and of jaundice.

Duration and Severity of the Disease and of Jaundice: This was taken to be the number of days which comprised the time from the onset of symptoms to the day of discharge from the hospital. This period varied from 21 to 124 days. The average duration of disease in 45 patients, in which it could be calculated with reasonable accuracy, was 52 days. In 17 cases, the disease lasted not more than 40 days, and in 16 the patients were still symptomatic by the 65th day of illness. By nature of our definition of duration of disease, it is implied that all these 45 patients recovered. Discharge of the patient from the hospital was determined as a rule by both clinical and laboratory signs of recovery with the exception of the Hanger's test. A positive Hanger's test was not considered an indication to hold the patient in the hospital, especially in the

presence of obvious clinical recovery, as manifested by disappearance of fever and jaundice, reversion of the liver to a normal state with reappearance of appetite and weight gain. The severity of the disease was judged on the basis of duration of jaundice, duration of the disease, and the level of bilirubin in the serum. We realize that there are other factors that could determine the degree of severity of the disease, but this classification appears to be the simplest, and perhaps the one that adapts itself best for analysis and comparison.

All the cases were treated under a similar regimen consisting of bed rest, high carbohydrate, high protein, low fat diet and multivitamins. To about half of them, infusions of glucose and amino acids were given during the first 10 days of illness. The cases that received the infusions did not have a shorter convalescence, but it seems that such additional treatment was given to those who appeared worse on admission. Hence, no valid conclusion can be derived.

In 40 patients in which accurate information could be obtained, the average duration of jaundice was 37 days, the range being from 15 to 80 days. No appreciable effect on its duration was observed in those who received glucose infusions. Age also had no apparent effect on the duration of the disease or of jaundice.

There were two deaths in this series and both occurred during the first admission. In one case, liver biopsy showed evidence of the cholangiolitic form of infectious hepatitis and cirrhosis of the liver, and in the other, autopsy findings revealed lesions of both infectious hepatitis and post-necrotic cirrhosis. There were no other associated diseases in these cases.

Coincidental diseases were mostly parasitic infestations, as would be expected in the Puerto Rican veterans. (See Table IV). Of the 7 cases where hookworm ova were recovered from the stools, only 3 had a duration of the disease and of jaundice longer than the average. The 3 cases with Schistosoma mansoni infestation did not exhibit any remarkable complications clinically or otherwise. The case with Strongyloides stercoralis presented a rather severe clinical course both in intensity and duration of the disease but it is highly questionable that the parasitic infestation had anything to do with it, as there was no diarrhea or generalized abdominal pains. This patient's hemoglobin was 100%. The case with rheumatoid arthritis had a moderately severe hepatitis with jaundice of 2½ months duration, but interestingly enough, without any joint improvement. Anemia apparently had no effect on the period of convalescence, as there were many cases with mild anemia whose duration of disease and of jaundice was not above the average.

TABLE IV

COINCIDENTAL DISEASES

Disease	Number of Cases
Uncinariasis	7
Schistosomiasis	3
Tonsillitis	2
Strongyloidiasis	1
Rheumatoid Arthritis	1
Appendicitis (operated)	1
Sinusitis	1
Epididymitis	1
Latent syphilis	1

Laboratory Findings:

White Blood Cell Count: Though it has been reported[1] that a leucopenia is seen initially in infectious hepatitis, our findings do not bear this out. Eleven cases showed counts of less than 5,000 but this finding did not have a direct relationship with the severity of the disease and was not necessarily observed in its initial stage either. Of the 54 cases, only five showed white blood cell counts of over 10,000. This leucocytosis could not be explained on the basis of associated complications. The remaining 38 cases had normal white cell counts during their course of illness. Although a relative lymphocytosis was observed in the majority of the cases, an absolute lymphocytosis of over 3000 was actually found in only seven patients, and even here it was of minimal degree.

Serum Bilirubin: Serum bilirubin and/or icterus indices were done, as a rule, once a week. Apparently, what determined the severity of the case, was not the height of the abnormal value but the duration. In other words, there were cases with very high bilirubin contents which turned out to be mild, while moderate severe or severe cases consistently showed a prolonged and persistent, though moderate, bilirubin elevation. The highest serum bilirubin observed was 15.9 mgs% and the highest icteric index 228 units, normal values being 1 mg% and 4-6 units respectively. Only 8 cases had more than 10 mgs% serum bilirubin and/or 100 units as the icteric index. It is of interest that the highest serum bilirubin levels in the patients who died were 3.2 and 7.1 mgs% respectively. Abnormal values were found as a rule early in the disease and correlated with the degree of jaundice. Though serum bilirubin and/or icteric in-

dex values were not the last tests to return to normal, they contributed heavily in determining the advisability for discharging the patient, when they became so. These two determinations correlated so well that their simultaneous performance, though frequently ordered in this series, did not appear to be necessary.

Cephalin-Flocculation Test: For this test to be considered positive, it had to be at least 2+ in 24 hours. On this basis, 42 cases showed abnormal tests, in 10 cases it was reported negative, and in 2 cases it was not performed. Of the 10 cases where the test was negative, it was performed on the 30th day of illness in three patients, in another three the date of illness was not known at the time that the test was performed, and two other cases were of the cholangiolotic form of infectious hepatitis. When the test was done within the first 15 days of illness it was positive in 11 out of 15 cases (73%). In the cases where it was done for the first time between the 16th and 25th day of illness, it was positive in 18 out of 22 cases (82%). Therefore, of 37 cases in which the test was done within the first 25 days of illness it was positive in 29 (80%). Thus, it is relatively good diagnostic test, but in regard to prognosis it does not appear to have much value. Eleven cases were discharged from the hospital in which the only abnormal finding was a positive Hanger's test, and none of these cases have had recurrences or complications to our knowledge. Moreover the degree of positivity of the Hanger's test does not appear to be a good index of the severity of the disease. There were nine mild cases with 4+ Hanger's tests and seven moderately severe to severe cases with negative to 2+values. As a matter of fact there are three cases where the Hanger's test became negative during the course of the disease. They all developed the characteristic features of the cholangiolitic forms as described by Watson and Hoffbauer[4] with a decrease in the cholesterol ester fraction, and a rise in the total cholesterol, icteric index, and alkaline phosphatase. Of these cases, two died, and the other had a moderately severe, prolongued course. Therefore, when the Hanger's test becomes negative in infectious hepatitis, it does not necessarily mean improvement, but at times may indicate progression into the more serious cholangiolitic form of the disease.

Thymol Turbidity Test: This test was performed in 20 cases during the course of the illness. It was abnormal in 18. This represents a 90% incidence. The normal value accepted was 4 units or less. This test had the highest incidence of positivity on a percentage basis. The two cases with a negative thymol turbidity test had a positive Hanger's. Fifteen of the 18 cases with a positive thymol test had a positive Hanger's at the same time as or before the thymol became positive. We were not able to confirm the re-

port[5] that the Hanger's test becomes positive before the thymol does, as in the majority of instances (15 out of 20 cases) when the patients were tested for the first time, both tests were reported positive. The patients in whom this test was carried out for the first time were already, at least, in the 3rd week of their disease. It is of interest that in the three cases of cholangiolotic hepatitis, at the time when the Hanger's test was reported negative, the thymol test was still positive. In those where specific information was available, there were four cases that had a positive thymol turbidity test and six who had a negative one on discharge. Two of the latter were obtained in the presence of a positive Hanger's test.

Alkaline Phosphatase: This determination was performed in 36 cases, 28 of which showed abnormal values for an incidence of 77%. The laboratory employed a modified Bodansky technique and values up to 7 units were considered normal. The highest value obtained was 33 units and this patient had the cholangiolitic form as proven by a liver biopsy. There were only three other cases, who had an alkaline phosphatase of over 20 units, one being associated with Laennec's cirrhosis and the other two being observed in patients with a severe form of the hepatocellular type of infectious hepatitis. Four cases had a normal alkaline phosphatase during their first two weeks of illness and all turned out to be mild, while 8 cases who had high alkaline phosphatase values close to the onset of the disease, turned out to be severe or moderately severe cases. Persistence of a high alkaline phosphatase did not always mean, clinically or by other laboratory methods, a severe case, or one going into the cholangiolotic form, for there were six cases that exhibited a high alkaline phosphatase till late in the disease, at a time when all clinical and laboratory data, with the exception of the Hanger's test, were indicative of recovery.

Total Serum Cholesterol and Cholesterol Esters Fraction: The normal total serum cholesterol in the VA Hospital laboratory is 150-250 mg%. The normal cholesterol ester fraction is accepted as being 65-75% of the total cholesterol. Serum cholesterol was done in 37 cases, eleven of which had elevated values, for an abnormal incidence of 30%. This differs from the findings of Gardner et al,[6] in whose series "true hypercholesterolemia was not found". There was no good correlation between a high cholesterol and the degree of severity of the disease. In so far as the cholesterol ester fraction is concerned, the value was abnormal in 25 of 35 cases where it was performed, for an incidence of 71%. This high incidence is in accord with the findings of Gardner et al.[6] In 18 cases where enough information was available to correlate the cholesterol ester fraction with the clinical course and/or other

laboratory tests, its degree of abnormality did not correlate with the severity of the disease. However, it was apparent that the patient's improvement, as manifested by a relatively rapid diminution of the serum bilirubin content, reduction in alkaline phosphatase, or beginning clinical recovery, was as a rule, preceded by a definite change towards normal of the cholesterol ester fraction. In this way, this test appears to be of prognostic value, for as long as this fraction remained subnormal, so did the patient. In contrast with the Hanger's test, which often remained positive when actual recovery of the patient was apparent, there was, as a rule, no discrepancy between the course of the patient and the cholesterol ester value, as both correlated well.

Bromsulphalein Test: This test was performed in 21 patients. Retention of the dye above 5% at the end of 30 minutes was considered abnormal. Of fifteen cases where the Bromsulphalein test was done near the time of discharge from the hospital, and when the patient was free from jaundice, seven showed an abnormal bromsulphalein retention in their serum one half hour after injection. One of these showed evidence of cirrhosis of the liver, and another had Schistosoma mansoni infestation. This incidence of abnormal BSP retention on recovery is in accord with the one reported by Capps and Barker[7].

Prothrombin Time: This test was done in 24 cases and it was found to be abnormal, according to the control, in only 8 instances, that is, in 33% of the cases studied.

Total Protein and A/G ratio: Of 32 cases where this determination was carried out, 23 had hyperglobulinemia, eight of which showed inverted A/G ratios. The latter include the three cases of proven cirrhosis of the liver. This represents an incidence of hyperglobulinemia of 72% and of inverted A/G ratios of 25%. Any globulin value above 2.5 gms% was considered abnormal. Nothing can be said as to the time when these values either became abnormal or returned to normal, as they were performed only once or twice at different stages of the disease in different patients.

Urine Urobilonogen: Though it is realized how important this determination is as a diagnostic test, no statement can be made on the basis of this series, since it was done in few cases and sporadically at best.

TABLE V

INCIDENCE OF ABNORMAL LABORATORY TESTS

Test	Normal Value	Number of cases where done	No. of cases where abnormal	Incidence in Per cent
Thymol Turbidity	<4 units	20	18	90
Hanger's	<2+ in 24 hours	52	42	80
Alkaline Phosphatase	<7 units	36	28	77
Hyper-globulinemia	<2.5 gm%	32	23	72*
Cholesterol Ester Fraction	65-75 %	35	25	71
BSP Retention at Discharge	<5% in 30 Min.	15	7	47
Prothrombin Time	Control Basis	24	8	33⅓
Total Serum Cholesterol	150-250 mgs%	37	11	30
Inverted AG Ratio	1-2	32	8	25*

* Includes 3 cases of proven cirrhosis of the liver.

GENERAL DISCUSSION AND SUMMARY

54 cases of infectious hepatitis have been analyzed. As previously reported,[8] this disease occurs with greater frequency in the younger age groups. Jaundice, anorexia, malaise, lassitude, dark urine and right upper quadrant tenderness occurred in over 80% of the cases. However, the prodromata consisted mainly of malaise, lassitude, anorexia and fever. Anorexia and malaise by themselves or either of them combined with another symptom, constituted the most frequent group of symptoms at the onset of illness.

The clinical picture was found to be similar to the one reported in the American literature[1,2,3,4,6] with the following exceptions: (a) Splenomegaly and lymphadenopathy were practically absent from our series. (b) Our incidence of light and/or clay colored stools was much higher. This may indicate a more marked involvement of

the intrahepatic bile duct system than that found in the ordinary case. In support of this statement are the facts that the average duration of jaundice in our series is 10 days higher than the one reported by Watson and Hoffbauer[4], that a marked elevation of the alkaline phosphatase was also a prominent finding, and that the total serum cholesterol was elevated oftener than reported. This difference was apparently not due to coincidental diseases, including Schistosoma mansoni infestation.

As a rule the symptoms that appeared first, disappeared first, with the exception of jaundice which usually was the last one to subside. When symptoms such as fever, anorexia, malaise and lassitude persisted for more than two weeks, the case usually turned out to be a severe one.

Only 3 cases showed spider angiomata, but these showed evidence of concomitant cirrhosis of the liver.

Severity of the disease was apparently determined more by the duration of the jaundice than by the height of the serum bilirubin content. It does not seem necessary to perform both the serum bilirubin and the icteric index in order to study a jaundiced patient. Either will suffice.

The thymol and cephalin-flocculation tests were the most accurate in diagnosis, but did not appear to have prognostic value in infectious hepatitis. A positive Hanger's test apparently is not an indication to maintain the patient hospitalized or in an inactive status, when all other clinical and laboratory signs indicate recovery. When the Hanger's test becomes negative during the course of infectious hepatitis, it does not necessarily suggest improvement, but at times, the progression of the condition into the more serious state of the cholangiolitic form.

The cholesterol ester fraction appears to have prognostic value, as the patient's earliest signs of clinical recovery were often preceded by a definite change in the percentage towards normal. As long as this fraction remained sub-normal, so did the patient.

The highest phosphatase value was obtained in a patient with the cholangiolitic form of infectious hepatitis. All the patients who showed a high alkaline phosphatase close to the onset of the disease, became severe or moderately severe cases.

Hyperglobulinemia was a prominent finding in this series, and abnormal retention of bromsulphalein at the time of discharge was not uncommon.

Of the 54 cases, two died. In one case, a liver biopsy showed evidence of the cholangiolitic form of infectious hepatitis and cirrhosis of the liver, and in the other, autopsy findings revealed lesions of both infectious hepatitis and postnecrotic cirrhosis.

Long term follow-up of this series of patients is not as yet available but it is contemplated.

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THE HEPATORENAL SYNDROME

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The clinical entity characterized by hepatic necrosis and associated renal insufficiency has been the subject of diverse studies in an attempt to explain its etiology. This syndrome has been reported following biliary surgery,[1,4] in crushing injuries of the liver,[2,5,6] following extensive burns, in toxemias of pregnancy,[7] associated with hepatotoxic agents,[8,9] and in biliary and hepatic diseases in general. Pytel in 1936[10] reproduced the hepatorenal syndrome by ligating the hepatic artery; Marangoni et al[11] have reported its presence in cases of the Waterhouse-Friderichsen syndromes; Schiff[12] reported primary liver carcinoma associated with oliguria, azotemia and terminal uremia.

It is therefore not infrequent to observe clinically the combined hepatic and renal damage which characterize this syndrome, even though the mechanism which produces it is not clearly understood. However, it is generally accepted that the hepatorenal syndrome is one of the large variety of disorders producing degeneration of the distal convoluted tubules of the kidneys, and which Lucke[13] grouped under the name of lower nephron nephrosis. It is not in the scope of this communication to discuss the various conditions which may give to a lower nephron nephrosis, some of which are: crushing injuries, transfusion reactions, sulfa intoxications, burns, chemical nephrosis (e.g. bichloride of mercury poisoning) and the hepatorenal syndrome. Ever since Lucke coined the term to describe the pathologic involvement of the lower nephron, an extensive literature has been devoted to it.

In 1913 Merklen introduced the term hepatorenal syndrome to point out the relative frequency of coincidental hepatic and renal failures. Reviewing the literature on the subject, what appears to be the cause for debate is the mechanism responsible for the renal damage; that is, whether it is caused by the precipitation of bile within the tubules in deeply jaundiced patients (bile nephrosis) or by the renal ischemia and anoxia produced by shock, or as a squela of the vasomotor collapse accompanying extensive liver necrosis. The vasomotor collapse is believed to be due to the accumulation of large amounts of toxins which can not be detoxified by a failing liver,[11,14] or to Shorr's vasodepressor material.[15] Shorr and his associates have demonstrated the presence of a vasodepressor substance, (VDM) identified as Ferratin, in the circulation

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of patients with decompensated cirrhosis of the liver or liver anoxia resulting from hypotensive states. A second enzyme system in the liver normally inactivates VDM, but deterioration of this VDM inactivation mechanism is precipitated by anoxia. Shorr and his co-workers have demonstrated that VDM or Ferratin, produces a marked antidiuretic effect when infused intravenously in dogs.

As Lichtman showed,[16] in cases with liver disease, renal manifestations occurred more frequently in the presence of jaundice, so that biliary nephrosis must be an important factor in the development of the hepatorenal syndrome. On the other hand, in cases where trauma to the liver, extensive burns, and the Waterhouse-Friderichsen syndrome have led to oliguria and uremia, the marked tubular lesions have been attributed to renal ischemia. Hence, a combination of factors is probably at play in most cases of this syndrome.

An interesting report by Marangoni and D'Agati,[11] published in 1944, described the presence of the hepatorenal syndrome in the Waterhouse-Friderichsen syndrome, the latter being characterized by acute hemorrhage into the adrenal glands during overwhelming septicemia, followed by shock and circulatory collapse. It was observed by the authors that if the patients survived the first stage of the Waterhouse-Friderichsen syndrome, a second or hepatorenal stage ensued, with oliguria and azotemia, and with pathologic findings of severe toxic hepatic necrosis and degenerative changes of the tubules and glomeruli. It is their feeling that the mechanism responsible for the appearance of the hepatorenal syndrome when adrenal hemorrhages occur is that following adrenal destruction the full brunt of detoxication falls on the liver and kidneys, already depressed by shock and toxemia. The failure of the liver to carry out its detoxifying function results in toxic hepatic necrosis and subsequently leads to the characteristic changes in the renal tubules and glomeruli.

It is proper to state now that originally it was believed by investigators that the glomeruli were spared in lower nephron nephrosis, a fact no longer considered wholly accurate.[17,19] It is true that the most marked degenerative changes are found in the ascending part of Henle's loop and in the distal convoluted tubules, but involvement of the proximal tubules and glomeruli may also be encountered.

CASE REPORT

J.T.C., a 53 year old white male, was admitted to the Medical Service of Damas Hospital, Ponce, P. R. on January 6, 1953, with a history of jaundice of two weeks duration. One week prior to

admission he had developed increasing lethargy and refused to eat. The patient, a chronic alcoholic, had been apparently well until two weeks before admission when he noticed that his sclerae and skin were yellow, and his urine dark in color. At the same time, he developed general lassitude and malaise, anorexia, swelling of his abdomen and legs, and pain in his right upper quadrant. Pruritus appeared in the week before hospitalization, together with lethargy and incoherent speech. His relatives stated that he was an alcoholic and that in recent weeks he had been drinking heavily, ever since his two sons had been sent overseas for military service. There was no history of past fatty food intolerance, previous viral hepatitis, Schistosoma mansoni infestation, ingestion of hepatotoxic agents or past renal disease.

On admission, the physical examination disclosed a well developed, fairly well nourished male, showing marked icterus. The temperature was 98.6°F, the pulse 88 and his respiratory rate 22. The blood pressure was 110/80. He was conscious and cooperative. The head and neck examination disclosed an icteric tinge to all mucosal surfaces, and a smooth, red tongue, suggestive of vitamin deficiency. There were many spider nevi on the chest. The heart and lungs were normal. The abdomen was distended with fluid; the liver was felt three finger-breaths below the costal cage and was tender; the spleen was not felt. There was 2 plus pitting edema of the legs.

Laboratory studies showed a red blood cell count of 4,570,000 with 90% hemoglobin, a white cell count of 32,600 with 88% polymorphonuclear leukocytes, 4% lymphocytes and 8% monocytes. Urinalysis showed a specific gravity of 1.008, a pH of 5.0 and microscopically, 3-5 coarse granular casts and 3-5 finely granular casts per high power field. The urine was negative for sugar and acetone, and positive for bilirubin. Blood serology was negative. Stool examination was negative, including the test for occult blood. Icterus index was 93.6; cephalin flocculation was 3 plus in 24 hours and 4 plus in 48 hours; the Van den Bergh test was directly positive with a total serum bilirubin of 21.6 mg.%. The alkaline phosphatase was 15 modified Bodansky units; the thymol turbidity 21 units; the prothrombin time was 19 seconds with a control of 12 seconds; total proteins were 5.1 gm.%, albumin being 1.1 gm.% and globulin 4.0 gm.%. The chest X-ray was negative.

Therapy consisted of a high carbohydrate, high protein, low fat and low salt diet, which was changed to a low protein diet as soon as renal failure was apparent. The patient was given multivitamin preparations, including vitamin K, crude liver extract hypodermically, and intravenous infusions of Amigen and dextrose in water.

He became increasingly toxic, delirious, and showed a marked

increase in jaundice. One week after admission, the icterus index was up to 105. His urinary output became scant, and laboratory studies showed a non protein nitrogen of 124 mg.%, urea nitrogen of 60 mg.% and a creatinine of 8 mg.%. The carbon dioxide combining power was 30 volumes %. An electrocardiogram showed evidences of myocardial damage but no suggestive findings of electrolyte inbalance (e.g. hyperkalemia).

Despite attempts to combat terminal uremia, the patient became comatose and died on the 22nd of January 1953, sixteen days after admission.

AUTOPSY

The body was that of a well developed and well nourished, markedly icteric man. The thoracic organs were found to be normal; no esophageal varices were found. Two thousand cubic centimeters of clear ascitic fluid were found in the abdominal cavity. The liver was found to be enlarged, weighing 2,350 gm. It was hard, nodular in appearance and green in color. The spleen was atrophic, weighing only 70 grams. It was also hard and granular and showed the same greenish pigmentation seen in the liver. The kidneys were both larger than normal, the right weighing 240 gm. and the left 280 gm. They showed marked congestion, were bile stained and had a soft consistency. The rest of the organs were normal. Microscopic findings: The significant findings were limited to the liver, spleen and kidneys. Liver: There was a marked increase in the periportal fibrous tissue, which not only formed bands between adjacent triads, and resulted in pseudolobule formation, but also infiltrated in irregular fashion between the cords of liver cells. There was a marked proliferation of small bile ducts. The hepatic cells showed advanced fatty changes and contained abundant bile pigment. There was much irregularity in the size of cells and in their nuclei. The bile canaliculi were often observed to be plugged with inspissated bile. Spleen: There was early fibrosis of the pulp, most marked just beneath the capsule. The reticuloendothelial system showed a diffuse hyperplesia, the pulp having numerous macrophages with a brownish green cast. Kidneys: The epithelium of the convoluted tubules exhibited numerous vacuoles which varied from finely foamy ones to large single ones which filled most of the cell. In other areas the epithelium showed only marked cloudy swelling, and a greenish-brown tinge. The tubules were filled with coagulated protein and numerous bile stained, hyaline casts, especially in the collecting tubules. The glomeruli were normal.

DISCUSSION

This case illustrates a rapidly downhill course in a chronic alcoholic with portal cirrhosis, in which the fatal termination was accelerated by a concomitant renal failure.

The cause of death in cirrhosis of the liver is usually gastrointestinal hemorrhage, as a result of bleeding esophageal varices in most instances, or hepatic coma. Douglas and Snell[20] in an analysis of 444 cases of cirrhosis of the liver found gastrointestinal hemorrhage to be the cause of death in 49.5% of the cases and hepatic insufficiency in 30.6%. The remaining cases of this series died of intercurrent infection, like bronchopneumonia and peritonitis, of unrelated causes, and of renal insufficiency. The case reported in this presentation is definitely one in which the rapid course leading to death was accelerated by the appearance of renal failure, as demonstrated by the laboratory evidence of uremia and the postmortem findings of portal cirrhosis and renal degenerative changes as described in the hepatorenal syndrome.

It is interesting to note that this case showed a leukocytosis of 32,600 white blood cells without evidence of intercurrent infection. This is in accord with other investigators who have observed a leukocytosis in cases with acute liver necrosis. Lucke,[21] in 125 fatal cases, observed in the majority of them a mild increase in the white blood cell count, although only five had counts higher than 15,000 and only one had 31,000. Snapper and Schaefer[14] reported on ten cases of acute yellow atrophy, all of which had a leukocytosis, with only two of them showing evidence of an intercurrent infection.

Several authors have noticed a normal or slightly increased blood creatinine in cases of lower nephron nephrosis[14,18] in spite of a marked increase in both the non protein nitrogen and urea nitrogen. This discrepancy is attributed to their feeling that creatinine is excreted mainly by the glomeruli and reabsorbed only minimally by the tubules, so that tubular damage alone, they believe, will not produce creatinine retention. This differs from the findings in the case reported in this paper, in which, despite glomeruli pathologically found normal, a high creatinine value was found.

A high non protein nitrogen value is present in many cases of hepatic cirrhosis. Hall et al[22] have reported abnormally high values in 208 out of 403 cases, for an incidence of 52%. Several factors are offered to explain this increase in non protein nitrogen determinations. In cases of advanced cirrhosis of the liver, there is usually terminal cachexia with increased breakdown of body proteins; if there is associated gastrointestinal hemorrhage, there

is blood protein digestion with a rise in non protein nitrogen; and lastly, there might be secondary renal insufficiency as illustrated by this case, with diminished urinary output and an abnormally high non protein nitrogen value. In the latter, the diagnosis is established by other evidences of renal failure, such as oliguria or anuria, uriniferous breath and stupor, acidosis, hyperpotassemia, and other characteristic signs of uremia.

The pathologic findings in this case showed the expected liver changes of Laennec's or portal cirrhosis, with hepatic nodularity and hardness, and microscopically by marked replacement of hepatic cells by fibrous tissue, pseudolobulation and bile duct proliferation. If classified according to Hall et al,[22] the liver of this case would have been included in their second or subchronic stage in which there is both fatty infiltration and portal fibrosis, as compared to their first or subacute stage in which the liver is very large and fibrosis is minimal (fatty stage) and the third or atrophic stage, characterized by the small, hobnail liver. In the latter there is very little, if any, fatty infiltration, fibrosis being the outstanding pathologic feature.

Lucke[21] reported that in the majority of cases of liver necrosis, at postmortem examination, the kidneys were swollen, flacid, and bile stained, a description which is in accord to the renal changes noted in these cases. Microscopically, degenerative changes were limited to the tubules in this patient, with findings of numerous vacuoles in the convoluted tubules and many bile stained casts in the collecting tubules, i.e. a cholemic nephrosis. The glomeruli were found to be normal.

In cirrhosis of the liver, the expected changes in the spleen, if any, are those of congestive splenomegaly, so often encountered in association with portal hypertension. However, occasionally, autopsy reveals atrophy of the spleen, as was the case in this patient. Its gross appearance showed marked contraction, a granular bile stained surface, and complete distortion of the normal architectural pattern. Microscopically, extensive fibrosis was encountered. Boyd[23] states that in the early stages of chronic congestion of the spleen there is increase in size, but that in long standing conditions, the spleen may become a fibrous organ. The reason for this fibrous change is not clear, but the accumulation of noxious agents in the organ, might be the etiologic factor involved. When the fibrous tissue shrinks, the spleen may become small and atrophic.

It is the general consensus of opinion that the management of the hepatorenal syndrome, as in all cases of lower nephron nephrosis, should be conservative. The successful treatment of two cases of the hepatorenal syndrome by exsanguinotransfusions

was reported in 1951 by Snapper and Schaefer,[14] but the merits of this method have apparently not been properly evaluated yet. Therefore, replacement of actual body fluid loss and careful observation for possible electrolyte inbalance is important. Hyperkalemia during the oliguric or anuric stage may occur rapidly and may cause death. Hence, accurate intake and output records, frequent laboratory determinations of non protein nitrogen, urea nitrogen, chlorides, potassium and carbon dioxide combining power, should be carried out. Repeated electrocardiograms should be performed to determine the presence of hyper or hypokalemia.

As is true in all cases of lower nephron nephrosis, in the hepatorenal syndrome, tubular regeneration starts after an average of ten days. The stage of spontaneous diuresis ensues at that time, and severely uremic patients often recover after being in a seemingly terminal condition. After the onset of diuresis, salt and water depletion and hypopotassemia should be prevented by an adequate fluid and potassium therapy. Unfortunately, the case presented failed to respond to therapy and died prior to this stage of diuresis.

SUMMARY

1—The report of a case of portal cirrhosis with jaundice and ascites who developed renal failure terminally is presented. The simultaneous occurrence of severe liver disease and kidney failure is described in the literature as the hepatorenal syndrome.

2—The pathologic and physiologic alterations encountered in the hepatorenal syndrome have been discussed. A variety of conditions that may lead to this clinical syndrome have been mentioned.

3—The clinical features of the case reported were analyzed. The pathologic findings in autopsy, consisting of portal cirrhosis, atrophy of the spleen, and biliary nephrosis were discussed. The renal lesions were found to be limited to the tubules, with sparing of the glomeruli.

The author is indebted to Dr. Mario R. García-Palmieri for his keen interest, valuable suggestions and cooperation in the preparation of this paper.

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FIBROSIS DIFUSA INTERSTICIAL IDIOPATICA DE LOS PULMONES

PEDRO J. DURAND, M.D.*

En 1944 Hamman y Rich presentaron los primeros 4 casos de lo que ellos llamaron **Fibrosis Difusa Intersticial Fulminante de los Pulmones.** Y la llamaron así porque todos sus casos murieron poco tiempo después del comienzo de la enfermedad. Desde esa fecha han seguido apareciendo en la literatura médica informes de casos bajo el mismo título hasta 1952 cuando ya se han reportado 14 casos en total.

El caso que presentamos hoy podemos decir que cuadra en esta nueva clasificación. Hasta donde puede saberse parece que es el primero relatado bajo este nombre en Puerto Rico y el tercero en la América Latina habiéndose informado 2 casos parecidos en Uruguay en 1948.

Los síntomas principales que presentan estos pacientes son los siguientes: tos, con cantidades variables de esputos, dolor en el pecho, hemoptisis ocasionalmente, fiebre, disnea progresiva y cianosis. Algunos casos han presentado dedos en forma de palillos en las manos y pies. La enfermedad comienza súbitamente en la gran mayoría de los enfermos, sin embargo, en otros ha surgido gradualmente. La duración de la enfermedad desde sus comienzos hasta la muerte también ha variado desde 34 días hasta 3½ años. Nuestro caso vivió 8 años.

Este es el caso de un hombre blanco, de 43 años de edad, veterano de la segunda guerra mundial, quien viene al hospital quejándose de disnea y cianosis. La historia presente comienza en 1944 en la ciudad de Nueva York cuando nuestro paciente desarrolla tos, fiebre, dolor en el pecho, y hemoptisis y es hospitalizado en el Hospital Mt. Sinai. Allí le dijeron que presentaba una sombra en su pulmón izquierdo y lo refieren en el término de dos semanas al Hospital Bellevue, ya que en dicho hospital sólo disponen de 12 camas para enfermedades del pecho y sólo hacen diagnósticos. En Bellevue el paciente permanece sólo una semana, con hemoptisis todo el tiempo. De allí lo refieren al Hospital Seton donde trataron de ponerle pneumotórax sin lograrlo. Permanece en dicha institución por 15 días con hemoptisis continuamente. Es transferido a Sunmount en Toper Lake. Allí le practican una interrupción del frénico en su lado izquierdo. Después de dicha operación el paciente comienza a experimentar disnea la cual continua progresivamente hasta su muerte 8 años más tarde. En febrero de 1946 es

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dado de alta de este hospital y se traslada al oeste de los E.U. donde está recluído en varios hospitales generales en California. En febrero de 1947, decide venirse a Puerto Rico. En noviembre de 1948 fué hospitalizado en San Patricio donde le practicaron una operación de mastoide.

Su historial médico no nos revela nada de importancia e interés. El paciente niega que haya estado expuesto a Bilharzia en Puerto Rico. No tiene historial de infecciones respiratorias recurrentes. No ha estado expuesto a polvos o gases irritantes. El era chofer y sirvió 10 meses en el ejército.

El examen físico nos revela un hombre de mediana edad en marcada dificultad respiratoria, con cianosis, ortopnea, y dedos en forma de palillo, en ambas manos. Presenta estertores finos inspiratorios y expiratorios regados en ambos lados del pecho. No se oyen estertores gruesos ni musicales. Nunca los tuvo durante sus 3 meses de hospitalización. Existe una preponderancia de respiración abdominal. La fase inspiratoria de la respiración estaba relativamente prolongada. Se notaba elevación del tórax durante la inspiración. Los sonidos respiratorios se oían distantes. Es notable la ausencia de hallazgos físicos en un paciente tan enfermo.

Resultado de los Exámenes del Laboratorio:

CR 4,550,000; Hbg. 86%; CB 8,950; Polis 62%; Linf. 34%; EOS. 4%. Indice de color 0.95; sedimentación corregida 40; Heces - negativa. Serología - negativa. Análisis de orina - negativa. Esputo - múltiples laminillas en el examen directo para B. K. fueron negativos. Exámenes bacteriológicos directos del esputo - técnica de Gram - negativo para hongos. Presentaba una mezcla abundante de estreptococos y cocos gram negativos. Cultivos - Sabourand y tioglicolato negativo para hongos. Cultivos - B.K. - negativos. Prueba de Tuberculina - negativa.

Consulta Cardiológica (Dr. E. Marchand)

E.K.G. fué negativo. No presentaba evidencia de cor pulmonale. No había evidencia de anomalía cardiovascular, para explicar el cuadro clínico. No había hipertensión pulmonar probada por la ausencia del cor pulmonale y P2 normal. Todo esto sugiere que la patología debe estar localizada en los alvéolos.

Curso de la Enfermedad y Discusión:

El paciente recibió tratamiento antibiótico, nebulizaciones, ACTH, oxígeno continuamente. Mejoraba poco temporeramente.

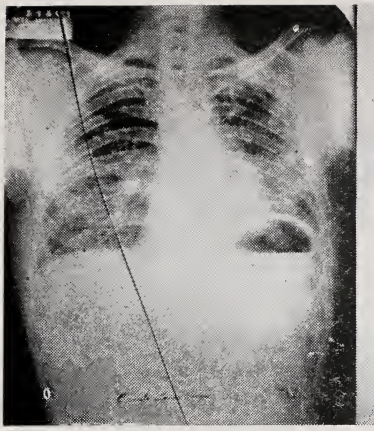

Fig. 1. Esta es la primera placa del paciente tomada al ingresar a la clínica. Revela una fibrosis generalizada en ambos campos pulmonares. El diafragma está marcadamente elevado en el lado izquierdo. Nótese la diferencia en el tamaño de los pulmones. El derecho pesó 620 gramos mientras que el izquierdo sólo 630.

Fig. 2 El peso de este corazón era de 150 gramos. Las cavidades ventriculares estaban contraídas y normal en tamaño. Las válvulas tienen un aspecto normal, normales en tamaño y anatómicamente competentes. *Ahora bien*, nótese la marcada hipertrofia ventricular derecha la cual media 1 cm. en espesor. La pared ventricular izquierda mide 1.5 cms. La hipertrofia ventricular se demuestra por el espesor de la pared. Si mide 5 mm. o más ya existe hipertrofia. En un estudio de 48 autopsias en casos de enfisema crónico y enfisema asociado con fibrosis, tuberculosis, o silicosis, informados por Scott y Garvin, encontraron que el ventrículo derecho media de 5 a 8 mm en espesor y casi todos los pacientes murieron antes que el ventrículo alcanzara un grueso mayor.

Fif. 3. Esta es una sección gruesa del pulmón. El parénquima ha sido reemplazado extensamente por múltiples espacios quísticos que le imparten al tejido pulmonar una apariencia de esponja o pan de abeja. Estos pequeños bolsillos de aire miden de 1 a 10 mm en diámetro. Los espacios son multiloculares.

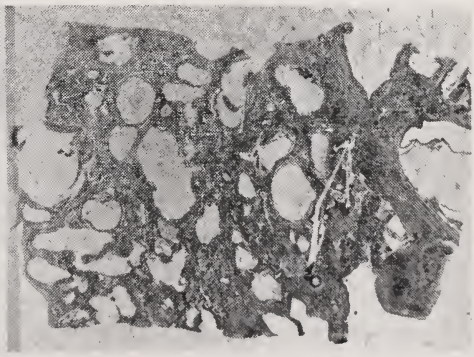

Fig. 4. Esta es una sección representativa del parénqima pulmonar demostrando las estructuras quísticas.

Siguió un curso progresivo hasta morir en julio 17 de 1952 con síntomas de fallo respiratorio. Murió por sofocación debido a la insuficiencia pulmonar, secundaria ésta a la destrucción de la elasticidad pulmonar y obliteración de la ventilación respiratoria alveolar.

La historia en este caso no demostró la presencia de enfermedad pulmonar anterior que diera lugar a fibrosis. no existió evidencia que pueda incriminar un agente bacteriano como un posible factor etiológico. Tuvo fiebre solamente durante el período agudo de la enfermedad sin repetirse más durante el curso de la misma. No presentó nunca inflamación supurativa del árbol tráqueo-bronquial. Estos hechos sugieren que la patogénesis de esta enfermedad no depende de una infección bacteriana anterior o concomitante. Durante la fase aguda de la enfermedad ocurre una progresión rápida de los síntomas, con disnea severa, cianosis y hemoptisis ocasionalmente.

Los cambios patológicos fundamentales consisten en un aumento en el tejido conectivo intersticial con impedimento de la corriente sanguínea en los pulmones. El desarrollo de edema intersticial y transudación de líquido a los alvéolos interviene con el intercambio de gases y es responsable de los síntomas clínicos de disnea y cianosis. El enfisema es el resultado de la hipernea y los esfuerzos respiratorios del paciente. Si esta condición persiste los alvéolos se dilatan y sus paredes se rompen. Según Kirshmer una presión de 100 a 300 mm. de agua presente en los alvéolos puede producir una rotura; y durante la tos la presión en el árbol bronquial puede alcanzar cifras tan altas como una tercera parte de la presión atmosférica, que es de 750 mm de mercurio. La unión de los alvéolos dilatados y rotos nos pueden dar grandes cavidades (bulas pulmonares). Cuando el aire se escapa a los tejidos intersticiales del pulmón (enfisema intersticial) puede localizarse en el tejido conectivo subpleural para formar ampollas pulmonares (blebs).

Este caso nos prueba que la fibrosis pulmonar no es siempre secundaria a infección como lo enfatizó Miller hace más de 20 años. La opinión hoy día es que muchos agentes etiológicos (hasta 75 se han mencionado) pueden provocar una proliferación fibroblástica en el tejido pulmonar. Esta reacción es la respuesta del tejido a la presencia de linfa en los espacios intersticiales asociada con la obstrucción crónica de la corriente linfática.

No nos ha sido posible determinar el factor etiológico específico en este caso. Un diagnóstico anatómico adecuado se ha podido hacer en este paciente con fibrosis pulmonar bilateral clínicamente indeterminada. Si considerarámos la lesión básica como inflamatoria debía ser el resultado de una infección por virus o irritación química. La primera posibilidad debe ser seriamente considerada. La segunda posibilidad fué casi eliminada por el historial.

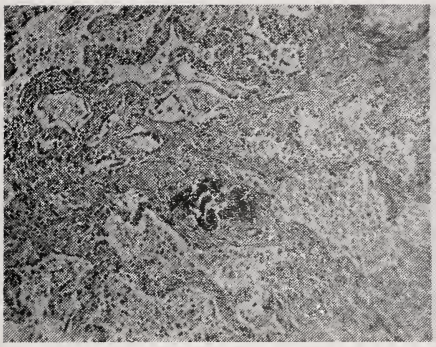

Fig. 5. Esta es una sección del parénquima demostrando los quistes más en detalle. Nótese la diversidad de tamaños, el parénquima está grandemente reducido, el dibujo vascular y bronquial desfigurado. Note materia albuminosa coagulada y células de congestión cardíaca en su lumen.

Fig. 6. Este es un espacio de aire con epitelio. Nótese la hipertrofia muscular.

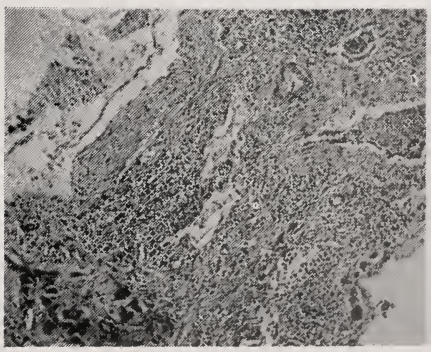

Fig. 7. Aquí se demuestra la fibrosis pulmonar e infiltrado de células redondas pequeñas, linfocitos, células de plasma y pocos eosinófilos y neutrófilos. El exudado celular está también presente en el estroma de la pared de los quistes, bronquiolos, y en un menor grado en los bronquios.

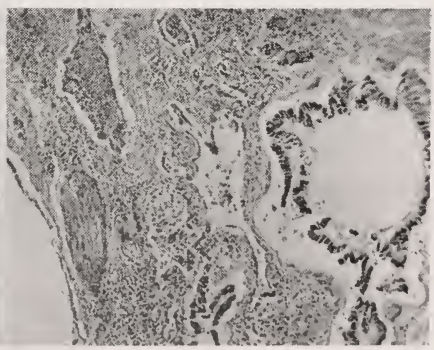

Fig. 8. Nótese quiste, bronquiolo, parénquima contraído y fibrosis.

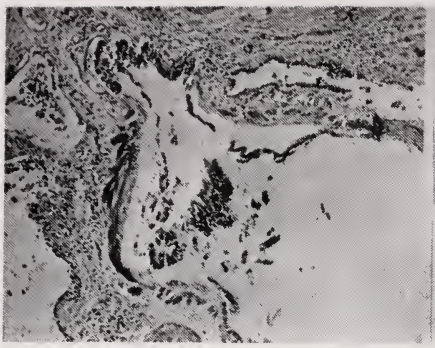

Fig. 9. Aquí podemos ver la comunicación directa de un bronquiolo terminal con un espacio quístico. Demuestra la hipertrofia marcada del músculo de la pared bronquiolar. En mi opinión si hubiera sido posible tomar cortes en diferentes planos y múltiples veríamos que todos estos espacios comunicaban con un bronquiolo terminal u otro espacio alveolar.

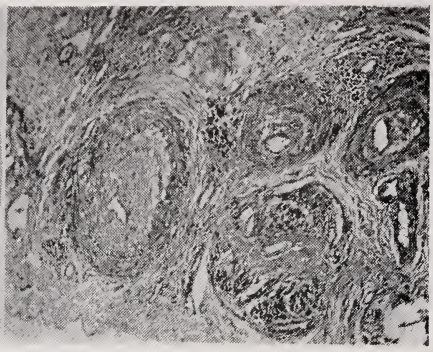

Fig. 10. Aquí aparecen las arterias pulmonares intrínsecas y arteriolos demostrando engrosamiento por esclerosis. La esclerosis vascular es característica de todas las formas de fibrosis pulmonar pero en este caso no es el factor predominante como ocurre en Ayerzas, Esquistosomiasis, estenosis mitral, embolismo y carcinomatosis metastásica.

CONCLUSION

Se presenta un caso de fibrosis difusa intersticial de los pulmones. La formación de múltiples bulas fué el resultado de la pérdida de elasticidad pulmonar y el esfuerzo respiratorio del paciente.

Sospechamos que esta condición puede tener varios factores etiológicos. Deseamos expresar nuestro agradecimiento al Dr. Félix Reyes por su excelente estudio histopatológico y al Dr. E. Marchand por su brillante consulta cardiológica.

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ROENTGENOLOGIC DEMONSTRATION OF ESOPHAGEAL VARICES*

LASZLO EHRLICH, M.D.

Esophageal varices were demonstrated for the first time roentgenologically by Wolf in 1928. The mechanism of production of esophageal varices is better understood by the studies of Kegaries, who injected the lower end of the esophagus and cardiac end of the stomach with india ink. He demonstrated that the veins of the lower end of the esophagus are very poorly supported by loose connective tissue, thus predisposing to the formation of the varices. In contrast to this, the veins of the region of the cardia are strongly supported and, having a rich plexus of anastomoses, offer a marked resistance to the flow of blood.

Portal hypertension producing esophageal varices is the result of blockage within the liver, as in cirrhosis, or extrahepatically as in the so-called Banti's syndrome. The blood is forced through the coronary and short gastric veins by retrograde pressure and enters the systemic circulation by way of esophageal anastomoses. Due to the poor support of the connective tissue in the lower end of the esophagus just mentioned, varices are produced. These veins are constantly exposed to trauma because the pinchcock action of the diaphragm and the corrosive effect of gastric acid in case of reflux; hence the occurrence of hemorrhages.

The demonstration of esophageal varices is of importance in:

1. Sudden and massive hematemesis, clinically obscure;
2. Persistent melena, with atypical G. I. symptoms;
3. Splenomegaly of unknown cause;
4. Hepatic cirrhosis, when liver and spleen are not palpable.

In all these instances the demonstration of esophageal varices indicate the presence of obstruction of the portal circulation and the potential danger of exsanguinating hematemeses.

In 115 cases of esophageal varices studied by Higgins, 61 died of hemorrhage. Only about half of them were diagnosed roentgenologically. Schatzki, who is particularly interested in this problem, also believes that half of the esophageal varices proved postmortem are not demonstrated roentgenologically. Doctor Hodes, on the other hand, in a personal communication stated that most of the esophageal varices can be demonstrated if strict adherence to the most careful technique is observed.

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* From the X-Rays Service of San Patricio V. A. Hospital.
Presented at the Annual Meeting of the Puerto Rico Medical Association, December 9-13, 1953.

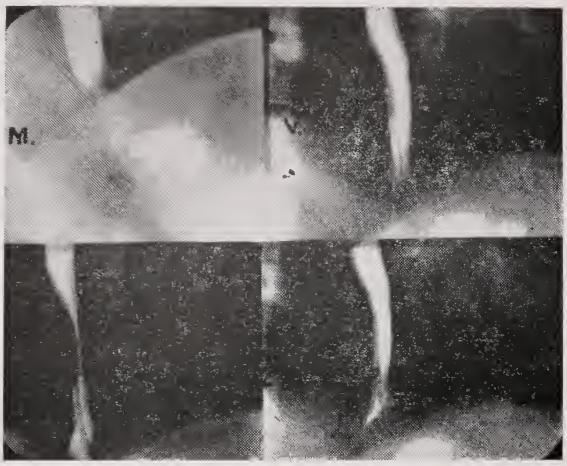

Fig. 1

Normally, the mucosal pattern of the esophagus appears as parallel, thin, longitudinal folds. (Fig. 1)

In the presence of varices, the normal mucosal pattern is markedly altered. The folds become thickened and tortuous. The bulging of the veins into the lumen creates a mottling which has been described as the "string of pearls" sign. This moth-eaten, wormy appearance is highly characteristic. (Fig. 2.)

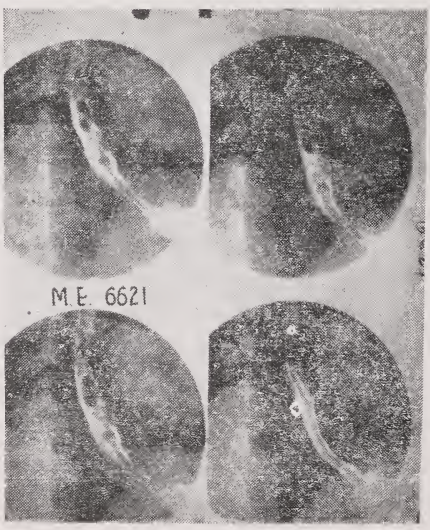

Fig. 2

Esophageal varices are almost always located in the lower end of the esophagus. They may extend upwards, involving the whole length of the esophagus. When they are extensive, their appearance is so typical that the diagnosis is obvious. When the varices are small or poorly demonstrated they can be missed.

From the point of view of differential diagnosis, the question of carcinoma should not arise, because the rigidity so characteristic of a malignancy is never present in esophageal varices.

Technique:

In my experience, the most satisfactory mixture for demonstration of esophageal varices is a fairly thick paste of barium, consisting of 2 parts of barium and one of water. Careful stiring is necessary in order to eliminate the presence of lumps or bubbles. (Fig. 3.)

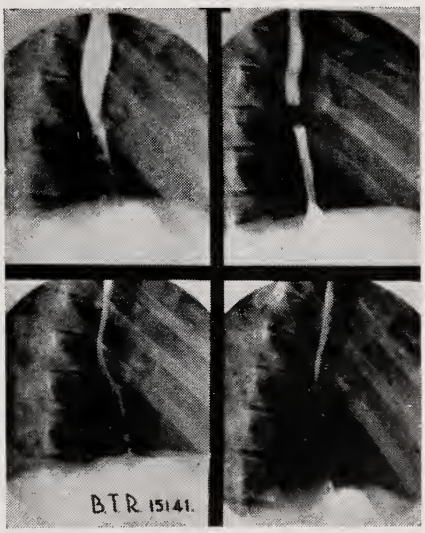

Fig. 3

The technique followed by us is essentially the same as the one used by Schatzki. One teaspoonful of barium mixture is administered to the patient in the recumbent position, face down, in a moderately right oblique position. After watching the descent of barium, the examination is particularly concentrated at the lower end of the esophagus. Spot films are taken when most of the barium has entered the stomach. Taking a film while the esophagus is full of barium is usually useless. Then spot films are taken while performing the Valsalva and Muller maneuvers.

In the Valsalva test, the patient attempts expiration after a deep inspiration, with glottis closed. In practice, he is asked to take a deep breath and then bear down.

In the Muller test, he takes a deep breath, expires forcefully and then tries to breath in, with his mouth closed and nose pinched.

In the Valsalva maneuver the intrathoracic pressure is increased and in the Muller maneuver it is decreased. Usually when varices are present, they are better visualized in one of these maneuvers. Theoretically, the varices should become larger during the Muller test, because of the increased negative pressure. Unfortunately, in practice, we find that most patients are unable to cooperate satisfactorily in performing these maneuvers. (Fig. 4.)

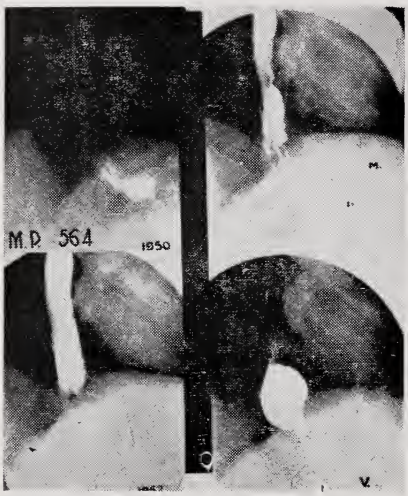

Fig. 4

If the examination in the prone position is negative, the patient is turned into the supine position, with his right shoulder up and the same procedure as above is repeated. Trendelenburg position may be helpful to slow down the passage of barium.

It is stressed that varices may not be seen during fluoroscopy. Spot films taken with correct coating of the esophageal mucosa and in different positions may show varices that were not visualized during fluoroscopy.

If the examination as outlined leaves the slightest doubt as to the presence of esophageal varices a second examination should be done. Usually repeated examinations can give a definite answer in most cases.

Radiographic examination without fluoroscopy is absolutely unreliable and therefore is condemned.

In San Patricio Hospital, in our small series of 17 cases of esophageal varices 8 were demonstrated roentgenologically, that is about 50%, on the basis of a single examination for each patient.

However, on repeated examinations, 15 were demonstrated out of 17, which is about 90%. (See Table)

In patient No. 7, 4 examinations were negative, and only on the 5th examination was the clinical diagnosis of esophageal varices confirmed.

Patient No. 17 had hematemesis, but his varices could not be demonstrated in spite of 5 "barium swallows". Still, the esophagoscopist could readily see them. Esophagoscopy is not done routinely at San Patricio Hospital. It is definitely indicated when all the roentgen studies are negative and the patient bleeds.

It is interesting to note that in patient No. 16, there is no clinical evidence of portal hypertension. (There is no enlargement of liver or spleen). Yet he was found to have esophageal varices on repeated X-ray examinations and was also confirmed by esophagoscopy. (Fig. 5.)

Fig. 5

I have presented to you a brief discussion on the roentgenologic demonstration of esophageal varices.

In conclusion, I wish to reemphasize the following points:

1. There is no "routine" technique for the roentgenologic demonstration of esophageal varices.
2. In the presence of a definite clinical suspicion, a single negative examination should not be accepted as final.
3. It is believed that repeated examinations, employing the most careful and individualized technique should succeed in demonstrating esophageal varices in a high percentage of cases.

TABLE I.

	PATIENT X-RAY NO.	AGE	HEMATEM.	HEPATO-MEGALY	SPLENO-MEGALY	ASCITES	FECES R. BIOPSY S. MANS.	LIVER F. TESTS H.	LIVER F. TESTS BSP.	BARIUM SW. SING.	BARIUM SW. REP.	SURG.	FINAL DIAGNOSIS	REMARKS
1	M. J. 2250	58	0	Not Palpable	4+	+	+	−	−	+		Splenect.	L. Cirrh. S. Mans.	
2	R. C. A. 15842	42	0	4+	4+	0	+	+		−	+	0	"	
3	M. R. 14148	26	+	1+	0	0	+	+	−	+	+	Refused	"	
4	[illegible] 2291	58	++	0	4+	0	+	−	−	−	+ +	0	"	Died
5	S. J. 7535	27	++	4+	4+	+	+	+	+	+		0	"	
6	[illegible] 5834	57	+	2+	4+	+	+	+		−		E-Gastro.	"	Died. PM
7	M. P. 564	48	+	4+	1+	+	+		+	−	− −+ −	Splenect.	"	
8	N. E. 8068	27	++	2+	2+	0	+	+	+	−	+	" E-Gastro.	"	
9	G. A. 10272	41	0	3+	4+	0	−	+	+	+		"	L. Cirrh	
10	M. J. 3538	56	0	2+	0	+	−		+	+		0	"	
11	R. CH. 15092	62	0	4+	2+	0	−	+		−	+	0	"	Died. PM
12	M. E. 6621	33	++	3+	4+	+	−	+	+	+	+ +	Refused	"	Blakemore used
13	C. M. 7495	55	++	4+	2+	0	−	+		+		0	"	"
14	R. C. 11593	56	+	0	2+	+	−	+		+		E-Gastro.	"	Died. PM
15	G. A. 4512	35	++	1+	Splenect. in 1944	0	−	+		−	− +	Subtot. Gastrect.	"	
16	S. J. 14126	64	+	0	0	0	−	−	−	+	+	0	E. Varix c. u.	Esophago-scopy: +
17	T. M. 2126	30	++	2+	0	0	−	+	−	−	−− −−	Refused	"	" ++

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THE REHABILITATION OF THE LARYNGECTOMIZED PATIENT

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During the past 7 years 24 veteran patients have been admitted to the San Patricio Hospital with the diagnosis of cancer of the larynx and hypopharynx. Out of these, 11 cases were considered suitable for total laryngectomy or laryngo-pharyngectomy, partial esophagectomy, radical neck dissection and partial or total glossectomy. One case was treated by laryngofissure. Seven of these operated patients are alive and present no evidence of cancer after periods ranging from one to 4½ years.

During the past month of June, three cases of operable cancer of the larynx in this hospital refused surgery and were treated by deep X-rays. The justified fear of losing their voices after surgery has cost the lives of many patients. We as physicians and oto-laryngologists must accept the responsibility of enlightening these unfortunate patients and taking fear out of their hearts. Every effort must be made to convince the patient that following surgery his voice can be very satisfactorily rehabilitated. The fact that not less than 30 articles on the rehabilitation of the laryngectomized patients have recently appeared in English, Spanish, French and Portuguese medical journals testifies to the growing interest on this subject.

Considering the tremendous psychic trauma in these cases it is proper to start well ahead of surgery with the psychologic preparation and encouragement necessary to accomplish the adjustment of the patient to the result of the operation.

There are three ways in which the laryngectomized patient may be rehabilitated:

1. The electrolarynx.
2. The Bell's artificial larynx.
3. The development of a bucopharyngeal or bucoesophageal voice.

The electrolarynx is a vibrating plastic disc encased in a cylinder and activated by an electric battery. The instrument is held against a selected site at the side of the neck while vibrating and the lips and mouth are moved as if in normal speech. After prolonged practice a very satisfactory artificial voice may be produced.

The Bell's artificial larynx is a simple device similar to a harmonica. The instrument is temporarily connected to the per-

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manent tracheotomy tube. During expiration a distinct audible sound is produced which is channeled into the mouth thru a mouthpiece where modulation into words is accomplished. A distinct monotone is thus produced. An illiterate individual who has undergone extensive neck surgery and X-ray therapy will probably handle a Bell's artificial larynx best.

Learning to use an artificial larynx is not like putting on a pair of new glasses and walking away. It takes a prolonged period of adjustment. We must watch our patient, lest he puts the artificial larynx under his pillow and forgets about it. We must convince him that it is not a disgrace to use an artificial larynx. It has been the experience in this hospital that very soon the apprehension and concern over the results of the operation are lost and the patient with his new voice becomes the center of attraction and all his fellow patients admire him.

There are a few little handicaps in using an artificial larynx. When the patient gets a chest cold the artificial larynx may get plugged with secretions and frequent cleansings are necessary to be able to produce a distinct sound. One of our patients tells us that he could not tell his girl friend a secret while in the theatre because he could not make his artificial larynx whisper. Another patient told us how the battery of his electrolarynx ran down in the middle of a lively conversation and he did not have a spare with him.

A good buco-esophageal or buco-pharyngeal voice naturally is the goal in the rehabilitation of the laryngectomized patient, but unfortunately it is not always feasible. A proper selection of the cases is most important. The will to talk, an emotional make-up, should be considered. Until very recently we were unable to secure the services of a trained speech therapist. We are very pleased to know that there is one well trained therapist in the Island, whose services are at present available to the laryngectomized veteran.

It is a good plan to orient and adjust the patient prior to surgery, discuss with him normal speech production and the development of his new voive.We have been fortunate to bring in one of our laryngectomyzed patients who has developed a good buco-pharyngeal voice and talk to other patients before and after surgery. He has convinced many patients who had refused surgery of the benefits of the operation. Thus he has saved several lives. He has been a real source of inspiration and encouragement to the patients as well as to us.

Mrs. Lois Senft, Supervisor of Speech Pathology at Walter Reed Army hospital, where one of our patients was sent for voice rehabilitation, writes us the following:

"All of the patients laryngectomized at this hospital are provided thorough orientation. This includes:

1. Discussion of physiological anatomical changes to be expected following surgery.
   a. Chance of breathing.
   b. Physical limitations.
   c. Etc.
2. Loss of speech.
3. Pseudo-voice orientation.
   a. Esophageal voice.
   b. Mechanical devices.
4. Psychological adjustment.
5. Showing a film "New Voices".
6. Play phonograph recordings of various stages of progress in the acquisition of esophageal voice.
7. A good esophageal voice user visits the patient.
8. Eructations are attempted by the patient prior to surgery.

We find that this thorough orientation greatly allays fear regarding the adjustment following the surgery.

As soon as the patient is medically ready to commence voice training following surgery he is seen individually for treatment. The first step is to "make a noise". A belch is demonstrated and encouraged with cokes or coffee or even water. We find that when we become too technical with a patient, he fails to reach the goal of making a noise. Only as a last resort do we outline a step by step procedure as follows:

1. Put lips together.
2. Lock air as you swallow until a thump is heard.
3. Push in with diaphragm.
4. Open mouth and speak.

Next the vowels are attempted with one belch per vowel. The eructation is extended to support 2, 3, 4, 5, & 6 syllables per intake. Volume, pitch variation, and quality are improved. Using the volume meter as a measuring device, we find that our superior esophageal voice users achieve a level of 60 db or conversational volume.

Froechels suggests an aspirate method of speaking. This we encourage following some success with the belching procedures outlined above. A quick inhalation with mouth open seems to provide the necessary 4 cc of air needed for 2-3 syllable words. This technique is hard to put across, I think.

The refinements of esophageal speech are attempted throughout the therapy. Extraneous intake and outgo noise is discouraged, as are facial contortions and other distracting movements".

Statistics show that 25% of all laryngectomized patients are

not suitable candidates for buco-pharyngeal speech. The factors which influence the ability to achieve a pseudo-voice are still under research and investigation. When a partial pharyngectomy and upper esophagectomy are performed the patient will, in all probability, not develop a pseudo-voice and an artificial larynx should be the choice. Almost every patient in whom the pharynx and esophagus have been preserved, should develop a good pseudo-voice. Unfortunately, many of our cases have been advanced cases necessitating more radical procedures such as radical neck dissection, partial esophagectomy and pharyngectomy and several plastic procedures to reconstruct the pharynx. These cases obviously are not suitable for the development of a pseudo-voice.

The laryngectomized patient in general has few handicaps once his voice is rehabilitated. Certainly he can not swim, or the water would quickly get into his lungs thru the permanent tracheotomy and cause violent coughing spasm which incapacitate even the best swimmer. Deaths produced in this manner have been reported among excellent swimmers. Lifting power and the glottic pressure at defecation are diminished or lost, hence constipation is frequent. The sensation of smell is totally lost because no air goes into the olfactory regions.

Let us remember that anyone who belches can develop a buco-pharyngeal voice after laryngectomy. Most of us are natural excellent Coca-Cola belchers. Some of us are probably good champagne belchers. At any rate, the belch is the beginning of a pseudo-voice.

May I stress again that any patient can be rehabilitated by one or more of the methods discussed. With a little patience, persistence and understanding from patients, physicians and voice therapists, the ultimate goal of a pseudo-voice will be usually achieved.

PRESENTATION OF CASES

Case I. C. M., age 56, had a total laryngectomy at San Patricio Hospital on June, 1949. He had received deep X-ray therapy previously. A large pharyngeal fistula resulted after surgery, which was closed by several plastic operations. Because of extensive surgery and previous radiation, the development of a pseudo-voice was out of the question. The patient is illiterate. He received instructions on the use of a Bell's artificial larynx which he handles very well, and is happy with it. Four and one-half years after surgery there is no evidence of carcinoma in his throat.

Case II. D. C., age 55, is an alert, intelligent and cooperating patient. A total laryngectomy was performed at San Patricio

Hospital on June 19, 1950, preserving all perilaryngeal muscles. From the very beginning the patient showed the will to talk and discarded an artificial larynx. He was transferred to the Speech Correction Center, Walter Reed Army Hospital, for speech rehabilitation. Upon his return to Puerto Rico three months later he had developed an excellent pseudo-voice. At present, three and one-half years after surgery, there is no evidence of recurrence or metastasis.

We have had the opportunity to observe this patient radiologically and endoscopically during phonation. A considerable dilatation of the esophagus is evident on X-rays but there is no difficulty during deglutition.

With the anterior commisure laryngoscope we have observed folds of the mucosa at the entrance of the esophagus. These vibrate and move very much like the vocal cords do during phonation. The esophagus acts as a reservoir of air just like the lungs and trachea do in normal phonation. In this altered physiology of phonation, according to Ferrari, the patient swallows air precisely at the beginning of inspiration, at which time the esophagus is subjected to negative intrathoracic pressure. Next the esophageal air is expelled thru the normal cricopharyngeal narrowing and the mucosal folds at the mouth of the esophagus during expiration.

Case III. C. M., age 67, suffered from epilepsy and carcinoma of the larynx with metastasis. He had a total laryngectomy and right radical neck dissection at San Patricio Hospital on April 18, 1951. Instruction on the development of a buco-pharyngeal voice was unsuccessful. This patient uses an electropharynx with satisfaction. Three years after surgery there is no evidence of carcinoma in his throat and no evidence of recurrent metastasis.

Case IV. C. J., age 61, had a laryngectomy, partial pharyngectomy, cervical esophagectomy, partial thyroidectomy, and radical neck dissection at San Patricio Hospital on October 10, 1952. Several plastic procedures were necessary to close the pharyngeal defect. This patient is not suitable for the development of a pseudo-voice. At present he is receiving instructions on the use of an artificial larynx.

Case V. N. A., age 61. A laryngectomy, partial pharyngectomy, and radical neck dissection were performed at San Patricio Hospital on August 25, 1953. There is a slight postoperative narrowing at the pharyngoesophageal anastomosis. The patient is being rehabilitated with an artificial larynx and will be referred to the speech therapist for further rehabilitation of his voice.

Case VI. C. E., age 65, had a laryngofissure for carcinoma of the right vocal cord on October 11, 1953, at San Patricio Hospital. A very hoarse laryngeal voice was present three weeks after operation. His voice in all probability, will be good and strong within the next two or three months. Laryngoscopic examination at present is entirely negative except for the operative scar in the right side of the larynx.

Case VII. L. F., age 65, had a laryngopharyngectomy, partial esophagectomy, and thyroidectomy, and a bilateral radical neck dissection at San Patricio Hospital last November 18. The postoperative course has been satisfactory and because of his extensive surgery this patient will probably use an artificial larynx best. He will be referred to the speech therapist for further rehabilitation.

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THE REHABILITATION OF THE HEMIPLEGIC VETERAN*

HERMAN J. FLAX, B.S., M.D., M.Sc. (Phys. Med.)**

Hemiplegia, stroke or apoplexy, by definition is a paralysis of one side of the body. It is also characterized at the onset by a disturbance of the sensorium, either stupor or coma prevailing.

The causes of hemiplegia, by far the most common neurological condition that physicians have to treat, are four, namely: 1—Hemorrhage, 2—Thrombosis, 3—Embolism and 4—Direct Trauma.

Cerebral hemorrhage is the usual cause of apoplexy in the elderly, who have high blood-pressure changes, arteriosclerosis, and involvement of the kidneys, chronic nephritis. Other important etiological factors are: syphilis of the cerebral arteries, lead or alcoholic intoxications and infectious diseases.

Thrombosis is the development of a plug or clot in a cerebral artery, formed by the coagulation of the blood, and remaining at the point of its origin. This type of stroke is also found in the aged, who have arteriosclerosis with damage to the intima of the arteries.

Embolism is found in a younger person and is the sudden blocking of a cerebral artery by a clot or obstruction, which has been brought to its place by the blood current. The source is the damaged valves of the heart, from the wall of the pulmonary vein or aorta, the result of rheumatic fever and bacterial endocarditis.

Traumatic hemiplegia, a common occurrence in war, is secondary to direct brain damage caused by sheer force, cranial fractures, gunshot wounds or complications of surgical procedures in the brain. The present study does not include these patients, as the young veterans admitted to San Patricio Veterans Administration Hospital attended special rehabilitation centers for the hemiplegia in the United States and were usually self-sufficient upon arrival in Puerto Rico.

Each of these conditions requires similar methods of rehabilitation. However, it is important for the physician to diagnose correctly the etiological factor, so as to select the proper time to begin treatment. Those patients suffering from a cerebral hemorrhage should wait at least three weeks before being allowed out of bed. Those with embolism or thrombosis may be permitted out of

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bed as soon as they have recovered from the loss of consciousness and are rational, provided there are no other constitutional contraindications. Those cases of hemiplegia in which the rehabilitation measures cannot keep up with the pathological process, as in cardiac failure, encephalomalacia and malignant hypertension, should be excluded from treatment.

Covalt[1] has pointed out that the physician, who sees a hemiplegic patient for the first time, may utilize two simple tests to determine whether or not he may walk again. "If the patient is able to move the arm on the affected side freely, there is every reason to believe he will be able to walk since the arm is usually more affected than the leg on the paralyzed side. If the patient is able to raise the affected leg an inch or two off the sheets while in a supine position, there should be sufficient muscle power remaining to permit him to walk again."

The purpose of a program of rehabilitation for the hemiplegic patient, according to Deaver[2], is as follows: "1—To prevent deformities, 2— To treat deformities if they occur, 3—To retrain the patient in ambulation and elevation, 4—To teach the patient to perform the activities of daily living and working with his affected arm and hand, 5— To retrain the affected arm and hand to its maximum capacity and 6— To treat the facial paralysis and speech disability if they are present".

"When a patient is accepted for rehabilitation", Rusk[3] suggests, "He is first given a careful physical examination. Next, a muscle test is given to determine the power of both affected and unaffected muscles. The range of motion of the joints is checked and recorded. He is also given the activities of daily living test."

In the Veterans Administration, these activities of daily living test consists of 154 items,[4] divided into eating, communication, hygiene, dressing locomotion, household and miscellaneous activities. The scores indicate the skill accomplished within a reasonable time and are recorded in the following manner:

0—Cannot be accomplished.
1—Can be accomplished with human aid.
2—Can be accomplished with adaptation of environment (low bed, special toilet seat, ramp, etc.)
3—Can be accomplished with the use of mechanical aids (splints, braces, prostheses, crutches, wheelchair, etc.).
4—Can be accomplished without aids, adaptation or assistance.

The initial test is scored in red, and the changes in condition are noted by inserting the data in the proper blank. Those blanks filled with 0 and 1 indicate the activities which the patient must learn to perform if he is to return to an independent life.

Careful evaluation must also be made of the mental status of

the hemiplegic patient. The patient must be orientated as to time, place and object, as well as to be able to retain instructions given him the day before or even an hour previously. It is obvious when learning ability is affected to the extent of loss of memory, rehabilitation is not feasible regardless of the return of motor power. On occasions, it may be advisable to employ the services of a psychologist or even a psychiatrist to evaluate the mental capacity of the patient. This is especially important if the family will not accept the diagnosis of cortical degeneration.

If treatment is started early there will be no complicating painful deformities that require prolonged physical therapy. While the patient is still in bed, a foot board will prevent foot drop. Sometimes, a posterior-leg splint will prove more practical. If a splint is used, care must be taken that a decubitus ulcer does not develop on the heel. However, this is readily cured by relieving the pressure of the heel against the splint or mattress, using a foam-rubber "do-nut". Sand bags are placed against the external border of the affected thigh and leg to prevent external rotation. In order to avert the abductor deformity of the shoulder, a pillow is wedged into the axilla. These deformities can also be avoided by having the physical therapist or a member of the family carry each joint of the involved and uninvolved sides through a complete range of motion twice daily. Quadriceps and gluteal muscle setting as well as proper breathing exercises are instituted to maintain muscle strength and aërate the lungs.

After the acute phase of the illness has passed, the patient is ready to begin more active rehabilitation measures. A tripod is suspended from an overhead (Balkan) frame, or a rope of braided bandage is attached to the foot of the bed. The patient is instructed to grasp the tripod or bandage, using the good hand to anchor the affected one, and pull himself up slowly into a sitting position. Sitting up in bed early will help reestablish balance and save the patient many days of practice when he begins to stand again.

If the patient is aphasic, speech therapy should be started as early as possible. This therapy should be given by or under the direction of a trained speech pathologist. If this is impossible, a teacher of speech from one of the local schools or an intelligent member of the family can be shown enough of the principles of speech retraining[5,6] to begin this program. Although the speech has been affected, the family should be made to understand that the intelligence of the patient has not deteriorated.

A system of pulleys[7] can be arranged over the head of the bed or attached to the Balkan frame, so that pulley therapy can be started while the patient is still in bed. The involved leg and arm can be tied to the pulley rope, and movements initiated with

the sound arm and leg. This procedure will prevent ankylosis and a painful shoulder joint as well as train the patient in reciprocal motion, which is essential in walking. Pulley therapy should be carried out for at least fifteen minutes, three times daily, continuously through the rehabilitation training period.

Ambulation should be attempted as soon as the patient has recovered some of his strength and is able to sit on the edge of the bed. At first, balancing is practiced in the standing position in parallel bars or using the backs of two kitchen chairs placed by the side of the bed. Next, still using the chairs, the patient should be taught heel-toe walking in a reciprocal gait pattern.

At times, a brace may be necessary to correct the foot drop. A short, double-bar brace with a 90 degree stop at the ankle joint will hold the toes up, and if a Y strap is attached to the lateral side of the shoe to buckle over the medial bar, supination or inversion of the foot will be corrected. This type of brace gives the patient confidence so that he will flex his hip and knee. Sometimes, it may be indispensable to use a long-leg brace because of quadriceps weakness. If the type is ordered, the knee should be unlocked by slipping the ring placed on the medial bar, since the patient can get to this side easier with his good hand. Approximately 50 per cent of the hemiplegic patients will require a brace.

The patient progresses to the use of crutches and the four-point gait. Then, he is taught a reciprocal gait pattern using a cane and finally without the cane. In the advanced stages of ambulation he is taught to climb stairs, cross roads, to get into a bus or automobile and outdoor walking.

The hand of the hemiplegia is the last to recover and in most cases does not recover completely. Ninety per cent of all hemiplegics can be taught ambulation,[8] but very few can be expected to regain full use of the involved hand even with prolonged muscle reeducation.

The exercise program[2, 8] for retraining the affected arm should be started while the patient is developing one arm skills with the unaffected arm. This program depends entirely on the patient, who should be continually encouraged by the physician. The physical therapist must work with the patient so that he understands each exercise, the purpose and the manner of performance in order to achieve the maximum results. The purpose of upper extremity exercises is to strengthen the muscles, prevent contractures and fibrosis of the joints and to facilitate the use of the hand in performing some of the activities encountered in daily living, such as holding a paper down while writing.

The fingers of a spastic hemiplegia are most difficult to reeducate for any useful purpose. If the dominant hand is paralyzed,

it is more advisable to train the uninvolved hand to perform the necessary hand and finger functions. Occasionally, a cook-up splint or a pan-cake splint combined with a wrist splint may be useful if the fingers are tightly flexed. Sometimes, for esthetic value and to prevent dependent edema of the hand and fingers, a sling is used. This is not encouraged as it interferes with reciprocal movements of the upper extremity, and the lack of use will promote fibrous ankylosis of the shoulder and finger joints.

Occupational therapy is of great value in retraining the upper extremity, because it combines exercise and retraining with interesting activity. Covalt and Yamshon[9, 10] found the use of the tonic neck reflexes valuable in the functional training of the hemiplegic arm. They demonstrated that the spastic hemiplegic patient can extend his elbow to a greater degree and with greater strength when the face is turned toward the involved side.

Training in handling self or self-care activities is an extremely important phase of rehabilitation of the hemiplegic since most of these patients do not recover all their functions completely. For those patients whose disability is permanent, there are a host of gadgets or self-help devices,[11] that will enable the patient to accomplish an activity by himself or with the least possible help from someone else. These objects should be given to the patient only as a last resort, when all other methods of functional retraining have failed. They are usually of the one-handed variety, such as a knife that cuts by rocking rather than sawing and a hand brush that fastens to the wall with suction cups.

The physician can offer many helpful hints that will make dressing easier. For example, use snaps on clothing instead of buttons or zippers. Neck-ties or bow-ties, that come already tied, can be clipped to the collar. Also, rubber shoe laces, that do not have to be tied, can be purchased.

Furthermore, a special one-handed typewriter and numerous one-handed telephone aids are on the market to assist in communication. Also, if a wheel chair is finally found to be essential, a one-arm drive with the side of the drive specified should be purchased instead of the ordinary two-wheel drive. These are only a few of the many self-help devices that can be employed to make life easier for the hemiplegic patient.

Review of Patients:

Eighteen patients were referred for rehabilitation of their hemiplegia to the Physical Medicine and Rehabilitation Service at San Patricio Veterans Administration Hospital from August 1, 1951 until July 31, 1953. No patient died while undergoing treatment or had a second cerebral vascular accident.

TABLE I

Pat. No.	Age	Days before PM & RS	C.V.A.	Stroke No.	PM & R elsewhere	Days in PM & RS	No. treats.	Days in Hosp. after PM & RS	A. D. L.								Speech	Last time worked	Remarks	
									Locomotion		Eating		Hygiene		Dressing					
									B	A	B	A	B	A	B	A				
1	64	[illegible]	[illegible]	1	No	25	[illegible]	25	0	0	0	0	0	0	0	0	N	8 yrs.	Signed Self out of Hosp.	1
2	31	92	H	1	No	15	11	32	4	4	4	4	4	4	4	4	N	5 mos.	Self Sufficient on admission.	2
3	58	9	H	1	No	50	33	75	0	4	0	4	0	4	0	4	N	8 yrs.	13 Hosps. in 2 yrs. for heart failure.	3
4	57	144	H	1	Yes	90	56	140	0	3	1	4	1	4	1	4	N	1 day	Thrombophlebitis R. leg, pulmonary embolism.	4
5	58	316	H	1	Yes	123	55	20	1	4	1	4	1	4	0	4	N	1 day	Contracted Achilles Tendon.	5
6	36	90	H	1	Yes	88	51	133	2	2	1	4	1	4	1	4	D	8 yrs.	Schistosomiasis treated.	6
7	57	8	T	1	No	153	91	153	0	2	0	1	0	1	0	1	A	3 yrs.	Recovered muscle power. Cortical degeneration.	7
8	62	51	H	1	No	79	47	79	0	3	0	4	0	4	0	4	A	1 day	Flaccid paralysis. Long leg brace.	8
9	75	4	T	3	No	8	7	9	0	4	0	4	0	4	0	4	N	20 yrs.	Oldest pat. Least difficulty.	9
10	67	6	T	1	No	36	23	37	0	4	0	4	0	4	0	4	N	1 yr.	Neurosyphilis.	10
11	57	182	H	1	No	22	14	11	1	4	4	4	4	4	4	4	D	1 day	A.W.O.L. Good result.	11
12	58	21	T	1	No	15	13	15	1	4	1	4	1	4	1	4	D	1 day	Second Stroke 6 wks. after discharge. Discharged rehabilitated, 41 days, 30 treats.	12
13	64	147	T	2	No	7	4	10	4	4	1	4	1	4	1	4	A	4 yrs.	Speech problem.	13
14	61	15	T	1	No	48	27	48	0	4	0	4	0	4	0	4	N	1 day	Chronic duodenal ulcer.	14
15	54	226	T	2	No	47	19	0	4	4	4	4	4	4	4	4	N	1 day	Coronary occlusion delayed P.M. & R.	15
16	68	6	T	1	No	7	5	25	4	4	4	4	4	4	4	4	N	1 day	Diabetes Mellitus.	16
17	60	21	E	1	No	46	24	107	0	0	0	0	0	0	0	0	A	15 yrs.	Coronary Occlusion. Suspended treatment	17
18	59	13	Tr	1	No	17	12	0	0	4	0	4	0	4	0	4	D	1 day	Secondary to surgery.	18

C.V.A. Symbols
T— Thrombosis
H— Hemorrhage
E— Embolism
Tr—Trauma

Speech Symbols
N— Normal
A— Aphasic
D— Dysarthria

A D L Symbols
0— Cannot be accomplished.
1— Can be accomplished with human aid.
2— Can be accomplished with adaptation of environment, (low bed, hand rail, etc.)
3— Can be accomplished with use of mechanical aids, (splints, braces, etc.)
4— Can be accomplished without aids, adaptations or assistance.

One patient (No. 1) signed himself out of the hospital before little more than bed activities were started. Only one patient (No. 17) suffered a setback while undergoing treatment. He was stricken with a coronary occlusion, while walking to the Physical Therapy Section, necessitating immediate curtailment of all activities. This patient was not a good risk from the beginning as he was hospitalized for treatment of an auricular fibrillation and myocardial damage from a previous coronary occlusion at the time of his stroke.

A third patient (No. 7) could not be rehabilitated because of widespread cortical degeneration, despite satisfactory return of motor power. This patient was admitted with a quadriparesis and aphasia the result of multiple cerebral thromboses. In a few weeks, the rightsided palsy cleared up, but his aphasia did not improve. Gradually, his left side became stronger, but the patient could not understand the purpose of treatment and could not remember from one minute to the next what was taught him. At the present time, he is convalescing in a Veterans Administration Contract Hospital, a wheel-chair invalid, the only patient unable to return to his home.

Except for these three patients, all the others have made a satisfactory return to normal life. It is true that none have returned to gainful employment, but they are still alive and participate in the decisions of their families and are no burden to their household. Any attempt to interest them in vocational rehabilitation was met with a negative response, and little could be done to change their attitude. This feeling, in part, may be due to their receiving a pension for physical disability. But, it is also true, that there are practically no opportunities for employment of elderly hemiplegic patients in Puerto Rico, considering their physical and environmental limitations. On the other hand, the young veterans with traumatic hemiparesis entered whole-heartedly into a program of Vocational Education and Rehabilitation.

CONCLUSION

A program for the rehabilitation of the hemiplegic patient is a simple and an effective one, but it must be followed judiciously to insure maximum benefit. With the cooperation of the patient and of the family, who must help carry out the rehabilitation methods under medical supervision, many of the complications, usually found in the untreated hemiplegic, can be avoided, and the return to a normal life facilitated.

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PSICOLOGIA CLINICA Y MEDICINA*

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Introducción:

El origen de la psicología clínica como campo de estudio científico se puede trazar a los estudios de Francis Galton sobre diferencias individuales.[4] Su desarrollo incluye las investigaciones extensas sobre diagnóstico y tratamiento de enfermedades mentales en los últimos veinte años, especialmente a raíz de la Segunda Guerra Mundial. Durante toda su historia la psicología clínica ha estado íntimamente ligada a la medicina,[10] y hoy en día el psicólogo clínico es parte integral del grupo psiquiátrico para el diagnóstico y tratamiento de alteraciones emocionales. Aunque la psicología clínica es una profesión joven, su prestigio tanto en los Estados Unidos como en otros países va en aumento. Debido a que su aparición en Puerto Rico es reciente, quizás sea prematuro predecir su futuro en la Isla. Este dependerá mayormente de su aceptación por la profesión médica y la contribución que los psicólogos clínicos puedan hacer a la medicina. El presente ensayo tiene como propósito considerar algunos de los aspectos en que estas dos profesiones pueden colaborar.

Louttit[6] dice que "el objetivo primario de la psicología clínica es el ajuste o reajuste satisfactorio del individuo a un medio ambiente." Para llevar a cabo este objetivo el psicólogo clínico trata de entender la conducta del individuo en términos de sus reacciones a la vida diaria, de entender el ambiente del individuo que se estudia, de encontrar las áreas de conflicto entre el individuo y su ambiente, y de ayudar al individuo a efectuar los cambios que sean necesarios para establecer una relación más satisfactoria con los que le rodean. Para esto el psicólogo debe: primero, entender al individuo y su ambiente; segundo, ayudar al individuo a manejar su ambiente; y tercero, mantenerse alerta para mejorar sus técnicas. En otras palabras, las funciones del psicólogo clínico son: diagnósticas, terapéuticas y de investigación científica.

Preparación académica:

Veamos cuál es la preparación académica y práctica del psicólogo clínico. Esta consiste de ocho años de estudios; es decir,

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además del bachillerato, se requieren cuatro años adicionales de estudios, uno de los cuales se dedica a un internado en un ambiente neuropsiquiátrico. Este internado da experiencia en el manejo práctico de casos, similar a los internados en medicina.[9] La preparación académica conduce al grado de Doctor en Filosofía. A los cinco años de experiencia clínica obtenida después del doctorado, el psicólogo clínico puede tomar los exámenes de la Junta Americana de examinadores en Psicología Profesional. Es deseable que los psicólogos sean miembros de la Asociación Americana de Psicología ya que esta organización tiene normas altas para la admisión de sus miembros y un código de ética que estará llamado a seguir el psicólogo.

La Práctica de la Psicología Clínica:

La primera función del psicólogo clínico (diagnóstico) se lleva a cabo por medio de entrevistas con fines de evaluar al paciente. Los psicólogos en general son mejor conocidos en el campo de la medición de la personalidad, inteligencia, etc., y es quizás por esto que sean frecuentemente llamados "psicómetras." Pero debemos subrayar que el uso de pruebas psicológicas no es la única técnica que utiliza el psicólogo clínico para el diagnóstico de alteraciones psicológicas. Las pruebas constituyen solamente un medio de observar al individuo en una situación específica bajo condiciones controladas y normalizadas. La experiencia clínica y destreza en la entrevista son muy importantes en el diagnóstico de dificultades intelectuales o emocionales. El psicólogo clínico está interesado en saber cuál es la capacidad del individuo para enfrentar los impactos de la vida diaria y no le interesa obtener un número de respuestas "correctas". Por ejemplo, si usásemos únicamente el valor numérico del cociente de inteligencia de una prueba para determinar la capacidad intelectual del individuo, equivaldría a tratar de diagnosticar una enfermedad a base del número de corpúsculos blancos que se encuentran en el contaje celular de una muestra de sangre. Tanto en la prueba de inteligencia como en el examen de laboratorio, lo que tiene valor son las implicaciones del cociente de inteligencia o del número de leucocitos en relación con el cuadro total de síntomas presentes. Por lo tanto, la interpretación de los resultados de una prueba psicológica es hecha a la luz del cuadro clínico de síntomas, del historial del caso, y de las observaciones generales hechas por el psicólogo durante la administración de las pruebas y la entrevista con el sujeto. Esta interpretación es de utilidad para determinar el funcionamiento intelectual y emocional de un individuo; para diferenciar entre disturbios psiquiátricos y neurológicos; para determinar la etiología y evaluar el pronóstico de un caso en particular, etc.

Los resultados de las observaciones que acabamos de mencionar son muy importantes para la segunda función del psicólogo clínico; a saber; la terapia. En cuanto al psicólogo clínico se refiere, el objettivo que persigue la psicoterapia es ayudar al individuo a entender los puntos de conflicto entre él y su ambiente, y a encontrar medios de reducir la tensión emocional que producen estos conflictos con el fin de que el individuo pueda llevar a cabo una vida feliz y productiva. Estos conflictos pueden deberse a alteraciones emocionales, dificultades de ajuste relacionadas con el ambiente, o pueden ser precipitadas por alguna enfermedad somática, deformidades físicas, etc. El psicólogo, en colaboración con el psiquiatra, trata de conseguir que el individuo modifique su actitud hacia sí mismo o hacia su ambiente para reducir las dificultades que impiden una relación satisfactoria entre el individuo y su ambiente. Por ejemplo, un individuo puede ser ayudado a comprender cómo y por qué ciertas características de su personalidad afectan sus relaciones con otros individuos, o cómo enfermedades somáticas le impiden establecer y mantener una relación efectiva con el ambiente.

Hemos dicho que el psicólogo clínico debe esforzarse en perfeccionar sus técnicas a través de la investigación científica. Este es un aspecto esencial en el desarrollo de toda profesión. Para lograr este desarrollo, el psicólogo debe mantenerse alerta para la investigación de nuevos problemas e ideas. Cada caso visto presenta situaciones nuevas que meritan la investigación científica por el psicólogo. Solamente así podrá éste mejorar su capacidad para ayudar a estos individuos. En virtud de su preparación el psicólogo clínico está en condiciones de llevar a cabo labor de investigación. Las investigaciones de Halstead y sus asociados en casos de lesiones orgánicas en el cerebro[5] e hipertensión esencial[7] ilustran lo que el psicólogo clínico puede hacer en el campo de la investigación científica.

El psicólogo clínico, en sus funciones clínicas, generalmente trabaja en colaboración con un psiquiatra y un trabajador social psiquiátrico como miembro de lo que comunmente se llama un grupo psiquiátrico.[8] Algunos grupos incluyen un número mayor de personas tales como neurólogos, enfermeras y otros especialistas. Todos los miembros del grupo estudian los casos que se les refieren y todos contribuyen a preparar un plan de tratamiento. Quien ha de manejar el caso para fines terapéuticos dependerá, en última instancia, de los requisitos del caso en particular, y de los factores que hayan predispuesto o precipitado la reacción emocional correspondiente.

El funcionamiento del grupo es responsabilidad del psiquiatra. Bajo su supervisión, el psicólogo clínico y los otros miembros del

grupo ejercen las funciones de diagnóstico, terapia, investigación, etc., que sean necesarias para lograr el restablecimiento rápido y eficaz del individuo. El grupo funciona generalmente en sitios tales como clínicas de higiene mental y hospitales psiquiátricos. El concepto del grupo es aplicable a la práctica privada y es de esperarse que cuando se refiera un caso al psicólogo clínico para fines terapéuticos, el caso habrá de verse en consulta con un psiquiatra y no por el psicólogo sólo.

El concepto del psicólogo clínico en la práctica privada es algo relativamente nuevo en el campo de la psicología. Su participación en psicoterapia ha sido motivo de preocupación tanto para la profesión médica[1] como para los psicólogos mismos.[3] Pero esta preocupación no existiría si se considerara que los esfuerzos terapéuticos del psicólogo clínico deben llevarse a cabo solamente bajo la égida de un psiquiatra. La duda en cuanto a la práctica de terapia por psicólogos clínicos surge solamente en aquellos casos en que el psicólogo se dedica a la práctica independiente de psicoterapia.[2]

Las funciones del psicólogo clínico pueden ser de mucho valor para los médicos en general, no importa cuál sea la especialidad del médico. Por ejemplo, ofrece al pediatra información importante para el diagnóstico y tratamiento de problemas de conducta y retardación mental en los niños; colabora con el especialista en medicina interna y el médico general en el tratamiento de enfermedades psicosomáticas tales como úlceras pépticas y dermatitis psicogénica; etc.

La Psicología Clínica en Puerto Rico:

Es temprano aún para evaluar la posición relativa de la psicología clínica en Puerto Rico. Hasta hace muy poco tiempo no había individuos debidamente preparados en este campo en la isla. El público en general desconoce todavía la existencia de esta profesión aún cuando se hace sentir su necesidad de tener personas con quien discutir sus problemas. Tanto las agencias públicas como las privadas están empleando mayor número de psicólogos en actividades de higiene mental y es de esperar que esta demanda aumente el número de personas que deseen entrar en esta profesión. Recientemente se ha fundado la Asociación de Psicólogos de Puerto Rico cuyo objetivo es mantener un alto nivel profesional en la práctica de la psicología.

Hemos tratado de dar a conocer a la clase médica de Puerto Rico algunos de los servicios que pueden prestar los miembros de una nueva profesión. Con el esfuerzo unido de ambas profesiones podremos lograr el objetivo común de llevar salud y tranquilidad mental a nuestro pueblo.

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* Board Diplomates

° Part Time

H or RO followed by * spends some time at Hospital and/or Regional Office.

PET MILK COMPANY, ARCADE BUILDING, ST. LOUIS 1, MISSOURI

Distribuidores: B. FERNANDEZ & HNOS., SUCRS.
San Juan, Puerto Rico

the ***most widely used***

ethical specialty for

care** of the **infant's skin

DESITIN®

OINTMENT

the pioneer external

cod liver oil therapy

Decisive studies[1,2] substantiate over 25 years of daily clinical use regarding the ability of Desitin Ointment to . . . . . *protect, soothe, dry and accelerate healing in...*

- **diaper rash** • **exanthema**
- **non-specific dermatoses**
- **intertrigo** • **prickly heat**
- **chafing** • **irritation**

(due to urine, excrement, chemicals or friction)

Desitin Ointment is a non-irritant blend of high grade, crude Norwegian cod liver oil (with its unsaturated fatty acids and high potency vitamins A and D in proper ratio for maximum efficacy), zinc oxide, talcum, petrolatum, and lanolin. Does not liquefy at body temperature and is not decomposed or washed away by secretions, exudate, urine or excrements. Dressings easily applied and painlessly removed.

Tubes of 1 oz., 2 oz., 4 oz., and 1 lb. jars

write for **samples** *and literature*

DESITIN CHEMICAL COMPANY

70 Ship Street • Providence 2, R. I.

1. Heimer, C. B., Grayzel, H. G. and Kramer, B.: Archives of Pediat. 68:382, 1951.
2. Behrman, H. T., Combes, F. C., Bobroff, A. and Leviticus, R.: Ind. Med. & Surg. 18:512, 1949.

Distribuidor:
COMERCIAL GODEL, INC.
Ave. Fernández Juncos 1608
Santurce, Puerto Rico

OCUSOL

a refreshing, soothing collyrium

FOR OCULAR IRRITATION DUE TO EYESTRAIN, DUST, SMOKE OR GLARE

Ocusol® is an isotonic, aqueous solution containing boric acid U.S.P. 1.1%, sodium borate U.S.P. 0.5%, berberine sulfate 0.01%, distilled extract of witch hazel N.F. 2.6%, camphor U.S.P. 0.04%, methylparaben U.S.P. 0.05%, rose oil 0.01%, glycerin U.S.P. 1.3%, NaCl U.S.P. 0.38% and water 94.01%.

Ocusol is harmless to the eyes; it may be used as often as required. Each package contains a sanitary, plastic eye cup.

Norwich

THE NORWICH PHARMACAL COMPANY

Norwich, New York, U. S. A.

Distribuidores: CESAR CASTILLO, INC., Edificio Camaleglo
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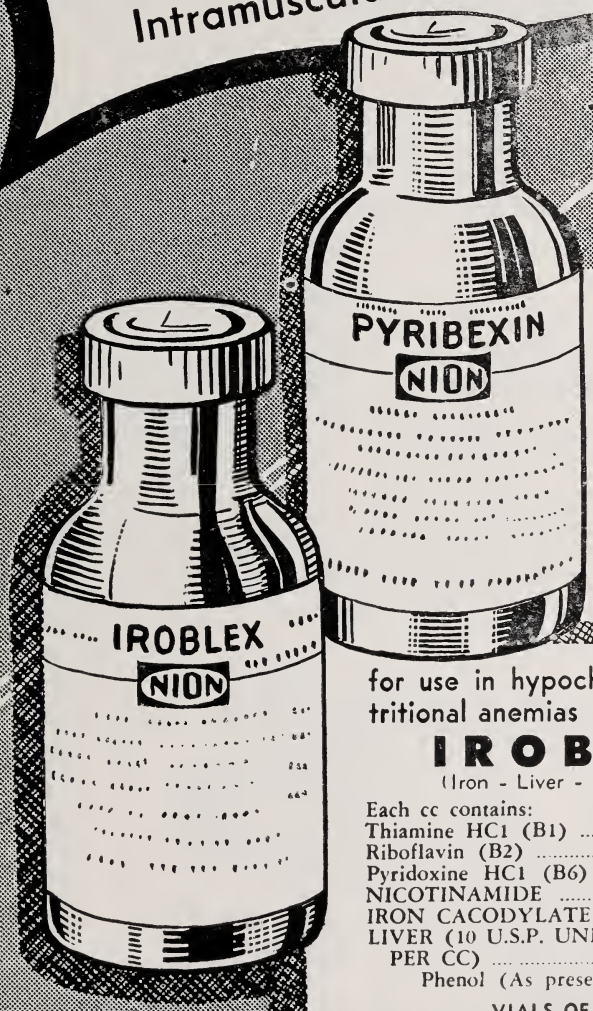
NION

INJECTABLE SOLUTIONS

Intramuscular . . . Intravenous



to relieve nausea and vomiting of pregnancy and in adolescent acne

PYRIBEXIN

(Pyridoxine HCl + Thiamine Chloride)

Each 1 cc contains:

Vitamin B1	50 mg.
Vitamin B6	50 mg.

VIALS OF 10 cc

for use in hypochromic and nutritional anemias

IROBLEX

(Iron - Liver - B Complex)

Each cc contains:

Thiamine HCl (B1)	100. mg.
Riboflavin (B2)	0.5 mg.
Pyridoxine HCl (B6)	1. mg.
NICOTINAMIDE	50. mg.
IRON CACODYLATE	10. mg.
LIVER (10 U.S.P. UNITS PER CC)	0.2 cc.

Phenol (As preservative) 0.5%

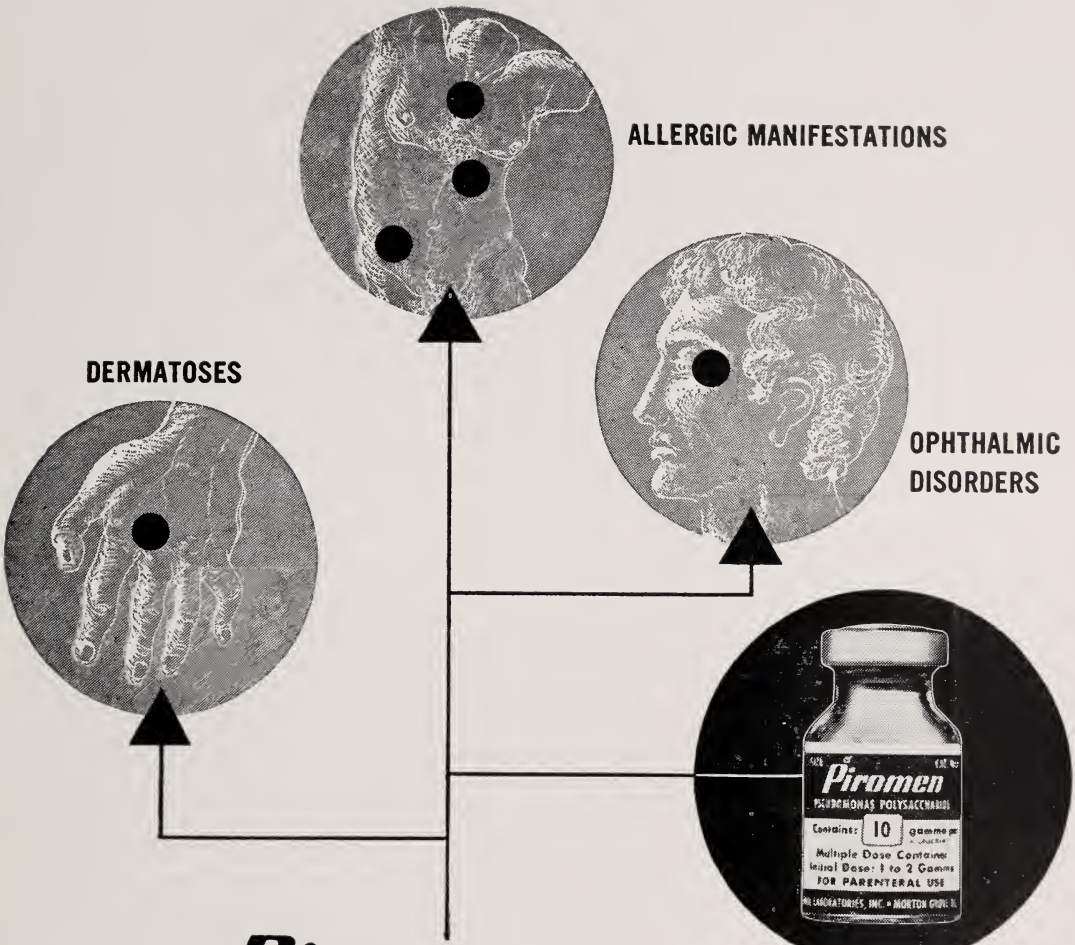
VIALS OF 10 cc

← Improved Formula

NION CORPORATION LOS ANGELES 38, CALIFORNIA

JOAQUIN BELENDEZ SOLA, INC.

P.O. BOX 1188, SAN JUAN, PUERTO RICO



Piromen®

(PSEUDOMONAS POLYSACCHARIDE) is proving effective in the control of a wide variety of allergies, dermatoses, and certain ophthalmic disorders. When injected, it produces a leucocytosis and initiates generalized activation of the reticulo-endothelial system. Supplied in 10 cc. vials containing either 4 or 10 gamma (micrograms) per cc., ***Piromen*** may be used safely within a wide dosage range.

For a comprehensive booklet detailing the use of this effective therapeutic agent, merely write ***"Piromen"*** *on your Rx, and mail to—*

Manufactured by **TRAVENOL LABORATORIES, INC.**

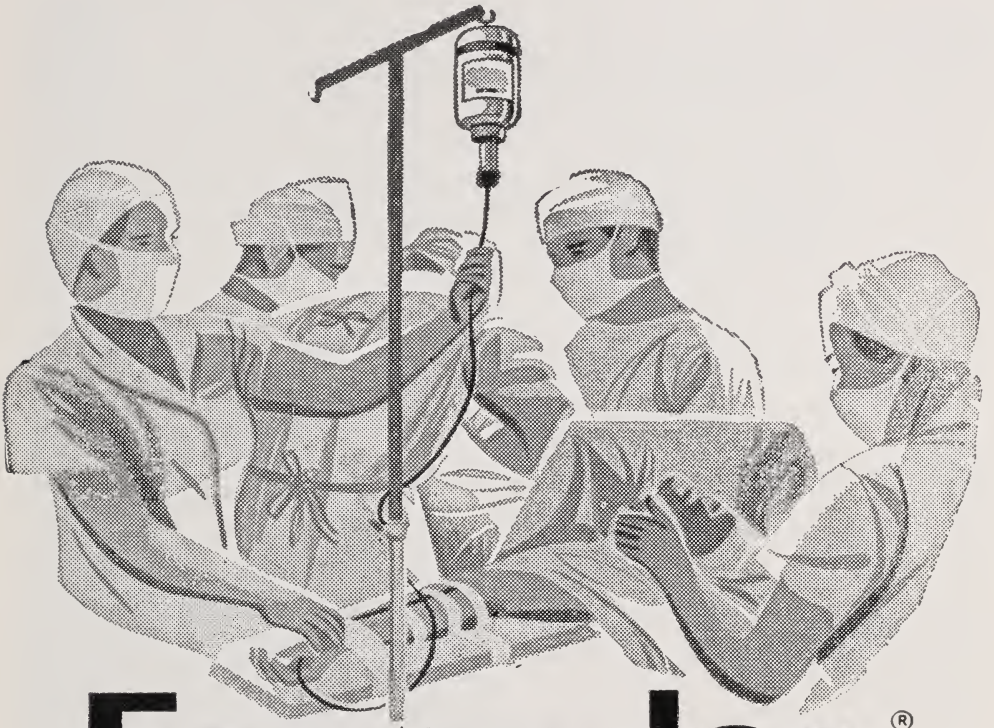
a subsidiary of BAXTER LABORATORIES, INC., MORTON GROVE, ILLINOIS

ASOCIACION MEDICA DE PUERTO RICO

ASAMBLEA ANUAL: DICIEMBRE 8-12, 1954

Anote esta fecha en su calendario de actividades médica.

AL PRIMER SIGNO DE COLAPSO CIRCULATORIO



Expandex®

PRODUCTO ACEPTADO POR EL CONSEJO DE FARMACIA
Y QUÍMICA DE LA ASOCIACIÓN MÉDICA AMERICANA

Restaura El Volumen Sanguíneo Circulante

Administrado al primer síntoma de descenso de la presión arterial, el Expandex impide los cambios irreversibles aumentando el volumen sanguíneo circulante y por lo tanto controlando el inminente colapso circulatorio.

Debido a su conveniente funcionamiento osmótico, el Expandex (6% de dextrán en solución salina isotónica) se emplea con éxito en el tratamiento y prevención del shock debido a hemorragias, quemaduras, traumatismos y cirugía. En la hemoragia puede usarse como el único líquido de reposición, siempre que la pérdida de sangre no exceda del 35 por ciento.

El Expandex es estéril, no es pirógeno y tampoco alergénico ni antigénico; no se almacena en cantidades importantes en el cuerpo. No es portador del virus de la hepatitis, y por consiguiente no la transmite. El Expandex es compatible con todos los grupos sanguíneos, no interfiere en la determinación de los mismos, su cruzamiento o del factor Rh, llevados a cabo posteriormente a su administración. Es líquido y se mantiene estable a temperatura ordinaria, por lo tanto viene listo para uso inmediato. No requiere nunca refrigeración.

La Inyección de Expandex, el primer producto de dextrán clínicamente aceptable elaborado en los Estados Unidos, se expende en botellas de 250 y 500 cc. Esta última se provee también empaquetada conjuntamente con el Equipo Estéril de Administración.

ACCEPTED COUNCIL ON PHARMACY AND CHEMISTRY AMERICAN MEDICAL ASSOCIATION

COMMERCIAL SOLVENTS CORPORATION

División de Exportación • 260 Madison Avenue, Nueva York 16, N. Y., E. U. A.

NEW! 4☆ star combination

for treatment of Infectious Diarrhea

brings ℞ profits and big volume

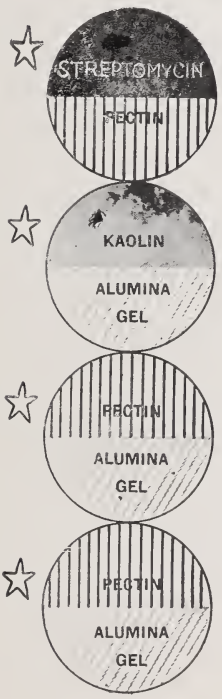
STREPTOMAGMA*

DIHYDROSTREPTOMYCIN SULFATE AND PECTIN WITH KAOLIN IN ALUMINA GEL

Special manufacturing techniques and rigorous controls, including repeated analyses, insure exceptional stability, smoothness of suspension and unusual palatability.

PROMPT, COMPLETE ANTIDIARRHEAL RESPONSE WITH

UNIQUE MULTIPLE SYNERGISTIC ACTION

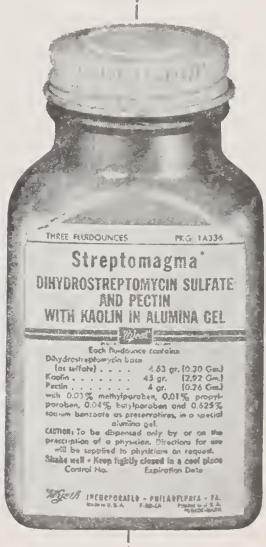


= BACTERIOSTASIS

= ADSORPTION OF TOXINS

= ABSORPTION OF WATER

= DEMULCENT ACTION



Streptomagma is supplied in bottles of 3 fluid ounces

Wyeth®

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This advertisement appears in the January, 1952 issues of:
American Druggist (7th)—Apothecary—Central Pharmaceutical Journal—Chain Store Age—Midwestern Druggist
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ILOTICINA

(ERITROMICINA, LILLY)

antibiótico

de eficacia

COMPROBADA CLINICAMENTE

CLINICAMENTE COMPROBADO... en el año que ha transcurrido desde el descubrimiento de la 'Iloticina' se han publicado más de 70 informes clínicos favorables. Los clínicos que han estudiado la 'Iloticina' en muchas partes del mundo en el tratamiento de una gran variedad de infecciones han comprobado el valor de este nuevo antibiótico.

La 'Iloticina' ha sido elogiada por su efecto sobresaliente contra muchos microorganismos sensibles a su acción, porque es BACTERICIDA contra muchos patógenos... porque es completamente eficaz contra muchos microorganismos resistentes a otros antibióticos... y porque tiene una baja incidencia de efectos secundarios.

'ILOTICINA'... la droga de elección en el tratamiento de muchas de las infecciones comunes.

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